Annals of Otology, Rhinology and Laryngology

FOUNDED BY JAMES PLEASANT PARKER

INCORPORATING

THE INDEX OF OTOLARYNGOLOGY

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ANNALS
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INCORPORATING THE INDEX OF OTOLARYNGOLOGY.

VOL. XXXV. MARCH, 1926. No. 1.

I.

AN OPERATION FOR THE REMOVAL OF THE SPHENOPALATINE GANGLION.

BY EDWARD CECIL SEWALL, M. D.,

STANFORD UNIVERSITY HOSPITAL,

SAN FRANCISCO.

The operation here described has been worked out by me upon the cadaver. I have never performed it upon the living. It is presented for consideration because I believe that newer methods of local anesthesia have brought this region into the surgical field with much hope of ultimate value.

Anatomy (Spalteholz).—Ganglion sphenopalatinum (Fig. No. 1) is a sympathetic ganglion and lies as a small, flattened wedge, with its apex behind, in the fossa pterygopalatina, just in front of the anterior opening of the canalis pterygoideus (Vidii), a few millimeters below and medianward from the n. maxillaris. The following are usually designated as its roots:

1. N. canalis pterygoidei (Vidii) enters the posterior apex of the ganglion, runs in the canalis pterygoideus (Vidii), there arising through the union of two threads:

(a) N. petrosus superficialis major (Fig. No. 2) comes off from the n. facialis at the ganglion geniculi, anastamoses with

the n. petrosus superficialis minor and arrives through the hiatus canalis facialis in the sulcus n. petrosi superficialis majoris of the temporal bone. It then passes lateral from the a. carotis interna through the fibrocartilago basalis, filling up the foramen lacerum, to the canalis pterygoideus. In it run certainly the sensory and perhaps also the taste fibers of the anterior two-thirds of the tongue; in addition it carries the secretory fibers for the lacrimal gland; for motor fibers which it is said to carry moreover see: n. palatinus posterior.

(b) N. petrosus profundus arises from the sympathetic plexus caroticus internus, woven around the a. carotis interna, from the horizontal part of the artery, and goes through the fibrocartilago basalis to the posterior opening of the canalis pterygoideus.

2. Fibers of the nn. sphenopalatini, which connect the ganglion with the n. maxillaris.

The following nerves are usually mentioned as branches of the ganglion, although they are, for the most part, direct branches of the nn. sphenopalatini, and are only strengthened by a variable number of threads from the ganglion.

1. Rami orbitales, two or three extremely fine threads which pass through the fissura orbitalis inferior into the orbital cavity and through the foramen ethmoidale posterius to the mucous membrane of the ethmoid cells and sphenoid sinus.

2. Rami nasales posteriores superiores, several fine threads, enter the nasal cavity through the foramen sphenopalatinum, and, as (a) rami laterales, arrive in the mucous membrane of the upper and middle nasal conchæ (some also through the canales pharyngeus and basipharyngeus to the fornix pharyngis and ostium pharyngeum tubæ auditivæ), as (b) rami mediales pass on the inferior surface of the body of the sphenoid bone to the nasal septum; of the latter, the largest, n. nasopalatinus (Scarpæ), extends forward and downward on the septum to the canalis incisivus, anastomoses with that of the other side and ends in the mucous membrane of the hard palate.

3. Rami nasales posteriores inferiores (laterales), usually branches of the n. palatinus anterior (vide infra), pass through

small openings in the pars perpendicularis oss. palatini into the nasal cavity and to the mucous membrane of the inferior concha.

4. Nn. palatini run downward through the canalis pterygopalatinus and the canales palatini, dividing into three branches:

(a) N. palatinus anterior, the largest, passes through the foramen palatinum majus on the inferior surface of the hard palate, passes forward on it, divided in several branches, and supplies the mucous membrane of the hard and soft palate and the gums.

(b) N. palatinus medius, the smallest, goes through a foramen palatinum minus to the mucous membrane of the soft palate and to the region of the tonsil.

(c) N. palatinus posterior goes through a foramen palatinum minus and then passes backward to the mucous membrane of the inferior surface of the soft palate. Some authors believe that it carries moreover to the m. levator veli palatini and m. uvulæ motor fibers, which are said to originate from the n. facialis and to pass through the n. petrosus superficialis major to the ganglion sphenopalatinum.

The fossa pterygopalatina (O. T. sphenomaxillary fossa) lies, deeply concealed, between the bones medianward from the fossa infratemporalis. It is united with the latter by a sickle shaped space, broad above, narrow below, which is closed below and lateralward by periosteum. Its medial wall is formed by the lateral surface of the pars perpendicularis oss. palatini, its upper wall by the anterior lateral portion of the inferior surface of the body of the sphenoid, the posterior (Fig. No. 3) by the sphenomaxillary surface of the ala magna oss. sphenoidalis and by the anterior surface of the processus pterygoideus, the anterior by the processus orbitalis oss. palatini and by the most posterior margin of the corpus maxillæ. Below it narrows to become the canalis pterygopalatinus, which is continued into the canalis palatini oss. palatini, and opens at the foramina palatina majus et minora on the under surface of the hard palate; in addition, delicate canals pass through the pars perpendicularis oss. palatini from the canalis pterygopalatinus into the nose. On the anterior wall connection is

made with the orbit by means of the fissura orbitalis inferior, with the anterior surface of the face by the sulcus and canalis infraorbitalis. On the medial surface, the wide foramen sphenopalatinum leads into the nose. From the posterior surface the foramen rotundum goes into the cavity of the skull, the canalis pterygoideus and the canalis pharyngeus to the lower surface of the base of the skull.

The anatomic descriptions of the pterygopalatine fossa fail to emphasize, as pointed out so clearly by Sluder, that the bones described as forming the walls are often completely hollowed out by the nasal accessory sinuses. The extensions of these nasal sinuses vary in different individuals, but the fossa may be separated anteriorly from the maxillary sinus by only a thin plate of bone, from the sphenoid sinus posteriorly, superiorly or even anterosuperiorly, and from the ethmoid cells anterosuperiorly by equally thin bony walls. Sluder also calls attention to the fact that the relationship of the ganglion to the nasal cavity itself is very intimate, it being in some cases practically submucous.

ARTERIES.

1. A. palatina descendens (O. T. posterior or descending palatine branch) passes downward to the canalis pterygopalatinus. Above it gives off the a. canalis pterygoidei (Vidii) (O. T. Vidian artery), which goes through the canalis pterygoideus backward to the roof of the pharynx. Below it divides into the aa. palatinæ minores et major, which run through the canales palatini and emerge from the foramina palatina minora et majus; the minor branches go to the soft palate and its surroundings, the major branch goes to the inferior surface of the hard palate as far forward as the gums.

2. A. sphenopalatina (O. T. sphenopalatine or nasopalatine artery), through the foramen sphenopalatinum into the nose; it supplies the lateral wall of the nose and the pharynx by means of the aa. nasales posteriores laterales, runs on the inferior surface of the body of the sphenoid to the nasal septum and breaks up into the aa. nasales posteriores septi, which anastomose in the canalis incisivus with the a. palatina major.

PHYSIOLOGY.

Sensations of touch, heat and cold and those due to chemical stimulants pass to the brain by way of the sensory fibers of the fifth.

Vasomotor control and secretory regulation comes from the sympathetic fibers. Some taste fibers make their way to the brain by way of the vidian.

PATHOLOGY.

While there is no histologic evidence to support it, the theory is tenable that changes in the ganglion, giving rise to various disturbances of function, may be caused by infection. In developing this theory it is logical to believe that the pathology may be due to direct inflammatory extension or to the effect from distant foci. The nose and accessory sinuses may furnish the septic foci to produce the ganglionic changes in either manner mentioned.

SYMPTOMS.

Certain complaints, some of them known for ages, have of late years been attributed to pathologic conditions of the sphenopalatine ganglion. The list of such aspersions is a long one, and it furnishes gratifying evidence of awakened interest in important structures, but is especially significant as a quickening in the realm of long neglected and distressing symptoms.

First under this characterization is to be mentioned headache. How little is known about this common and distressing complaint. What a step in advance if one type of headache can always be referred to the sphenopalatine ganglion. The headache that accompanies sinus infection, acute or chronic, is well known. It varies considerably in its distribution. I believe it is possible to have pain of frontal type in suppurative disease in the maxillary sinus. Also there seems to be a type of headache due to sphenoid or ethmosphenoid infection.

There are too many branches of different nerves to be taken into consideration for me to feel that I can diagnosticate with surety the one involved. I welcome, however, the work of Dr. Sluder and others, who have given so much pains to the clinical side of the question and who feel that they can distinguish headache as due to involvement of the sphenopalatine ganglion.

In my own experience, I have seen disturbances of the eye of trophic character definitely due to sinus disease. It has been established that disease of the optic nerve can be caused by sinus infection. It is also practically assured that trophic disturbances leading to corneal ulceration may be due to sinus disease. We naturally infer that such changes are due to the nerve involvement. Through what path the infection has traveled is a mystery, but the possibility of the irritation of the sphenopalatine ganglion must be considered. Again, the ethmoid nerves are so intimately associated with possibly diseased areas that they must be duly accredited as trouble makers.

DIAGNOSIS.

If repeated cocainization of the sphenopalatine ganglion causes cessation of the headache, or if injection of the ganglion by alcohol or carbolic acid gives relief, the relationship between the headache and the ganglion can be established.

TREATMENT.

Injection of the ganglion has given relief in certain cases, but the ultimate result has been disappointing. The effect of the injection is usually temporary, depending upon whether the injected material is placed directly into the substance of the ganglion. These injections may be made through the nose, following the technic of Dr. Sluder.

After the ganglion has been exposed, according to the surgical approach described hereafter, it can be injected with much more certainty. If injection is thought inadequate, the ganglion can be destroyed by the actual cautery, chemical cautery or some form of electric escharotic. Where such efforts have failed to relieve, or where a clean surgical procedure seems wiser, the following described technic will serve for the complete extirpation of the sphenopalatine ganglion:

OPERATION.

One to one and a half hours before the operation the patient is given hypodermatically scopolamin hydrobromid, gr. 1/150, and morphin sulphat, gr. ¼. On coming to the operating room he is placed on the operating table with head slightly raised. The parts of the face about the operative field are painted with

alcohol-iodin solution. The interior of the nose on the side to be operated on is painted freely with crystals of cocain and adrenalin; special attention is given to the region of the sphenopalatine foramen. A one per cent solution of novocain with adrenalin is injected under the upper lip at the gingivolabial junction and the needle is then passed upward over the anterior surface of the superior maxilla. The usual incision at the gingivolabial junction, used in doing a radical antrum operation, is made. There is usually very little or no bleeding under local anesthesia. See Fig. No. 4.

The periosteum and overlying cheek are raised from the bone and retracted and the antrum opened by chisel in the usual manner. There is usually no pain, but if the antrum appears sensitive, it is swabbed with cocain. Also at this time, or later, after the posterior wall of the antrum is broken down, the sphenopalatine ganglion may be injected with novocain for the purpose of anesthesia or with escharotics in case its removal is not desired. See Fig. No. V.

The mucous membrane lining the posterior wall of the antrum is now cut and dissected downward, as shown in Fig. No. VI. This flap of mucous membrane may be replaced after the operation is finished if deemed advisable.

The posterior wall of the antrum lying directly in front of the pterygopalatine fosas is now cracked by light blows of the chisel. The broken fragments are separated from the underlying periosteum and pulled forward with a narrow hooked-end spatula. The posterior antral wall is easily removed. See Fig. No. VII. The tissues occupying the pterygopalatine fossa are now exposed and present an undifferentiated mass. No effort is made to dissect the ganglion from the surrounding tissue. The whole mass containing the nerve branches and the blood vessels is removed as a unit.

The most confusing part of the operation now presents itself. If vessels are injured accidentally, bleeding would interfere with proper orientation. The method employed is the general surgical principle of keeping outside the periosteum and ligating vessels in turn as they are cut. Wth a narrow "submucous" spatula the periosteum is separated from the nasal wall of the pterygopalatine fossa. The dissector passes through the opening in the posterior wall of the antrum and hugs the nasal

wall much as in doing a submucous resection. The periosteum is thus removed from the vertical process of the palate bone, and the dissector then encounters the base of the pterygoid process, which forms the posterior wall of the pterygopalatine fossa. The dissection should first be kept below the branches of vessels and nerves that enter the nose at the sphenopalatine foramen. By working downward the palatine vessels and nerves can be raised and surrounded where they enter the pterygopalatine canal.

The first step has been attained in the recognition of the structures to be removed. The dissection is now carried upward, still hugging the palatine bone until the lower part of the bundle containing nasal branches is reached. The dissector now is moved upward to the sphenoid bone, and the periosteum is dissected downward, always working against the bone. The nasal branches now outlined from above and from below can be surrounded by a hooked instrument without damage. This represents the second stage in the identification of the structures to be removed. See Fig. No. VIII.

The lower bundle containing the n. palatine and the art. palatina descendens is now grasped in a delicate pair of artery forceps, cut off and a ligature thrown around by means of another pair of the same type of forceps. See Fig. No. IX. For those not familiar with this method of applying a ligature the technic is described as follows: (We use it to stop all bleeding after tonsillectomy, under local or general anesthesia, or whenever the ligature is to be tied in a position difficult of access.) The vessel is grasped by the hemostat as lightly as possible; a ligature is now knotted over this hemostat with one short and one long end. The short end must have the direction in which it is to be pulled. The short end is grasped close to the loose knot by means of a second hemostat, and the noose is pushed down off the end of the hemostat on the vessel, the long end of the ligature sliding without traction through the fingers until the knot is off the hemostat. Traction is then made on both ends, the lower by means of the forceps and the upper by the hand holding the hemostat.

The bundle entering the nose through the sphenopalatine foramen is now grasped, cut and tied. See Fig. No. X. The mass is now retracted by means of the two ligatures, dissected

free from its bed and the pterygoid process. The canalis pterygoideus (Vidii) comes now into view, the n. canalis pterygoideus and the a. canalis pterygoidei presenting and running forward to the ganglion. This bundle is hooked up, cut and tied off. See Figs. No. XI and No. XII.

The mass containing the ganglion, now well marked by the vidian, is grasped and ligated external to the ganglion. It is then removed by making traction on the ligatures and separating with a knife. See Fig. No. XIII.

Should there be disturbing bleeding it can be controlled by cauterization or packing, in case the ligatures have failed.

The flap of mucous membrane of the antrum may now be reapplied to cover the fossa. The wound at the lip is sewed in the usual manner.

NOTE: Since reading this paper at the meeting of the Western Section of the American Laryngological, Otological and Rhinological Society, in Los Angeles, in February, 1926, I have been gratified by having one of my colleagues here use this approach in operating on a tumor occupying the pterygopalatine fossa and entering the nose.

Doctor Sluder has kindly called my attention to work done by Carnochan along this line. Doctor R. J. Payne has sent me a reprint commenting on this work. I quote from Doctor Payne's article: "The earliest observations recorded in literature on the influence produced by lesions of the nasal (sphenopalatine-Meckel's) ganglion appear in the American Journal of Medical Sciences in an article written in 1856 by J. M. Carnochan entitled 'Exsection of the Trunk of the Second Branch of the Fifth Pair of Nerves Beyond the Ganglion of Meckel's for severe neuralgia of the face.' (Tic douloureux.) The author says 'the second branch of the fifth was exposed at the infraorbital foramen and followed along the roof of the antrum and floor of the orbit to the sphenopalatine ganglion, the nerve with the ganglion being removed en masse.'"

However, I can find no article on Carnochan in the American Journal of Medical Science, 1856, and have written for further help. I have not had time to learn more before it has become necessary for me to return this proof for publication.

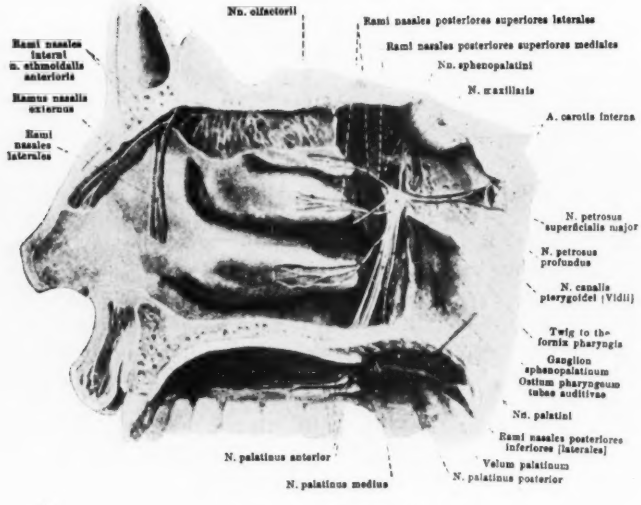

Figure 1 (Spalteholz).

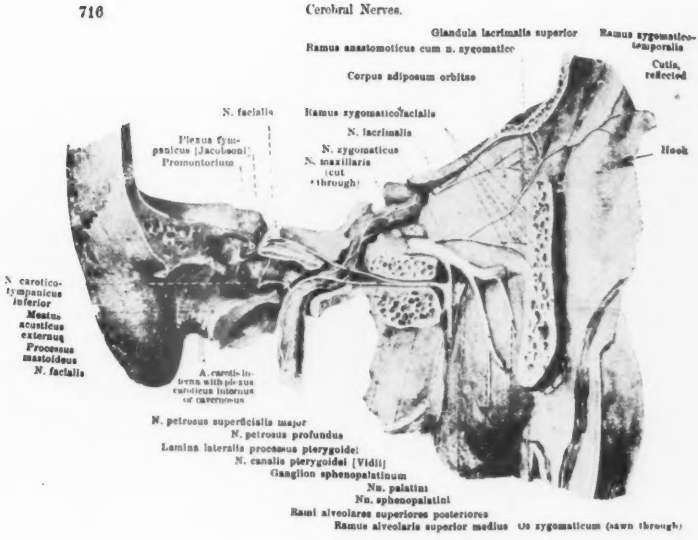

Figure 2 (Spalteholz).

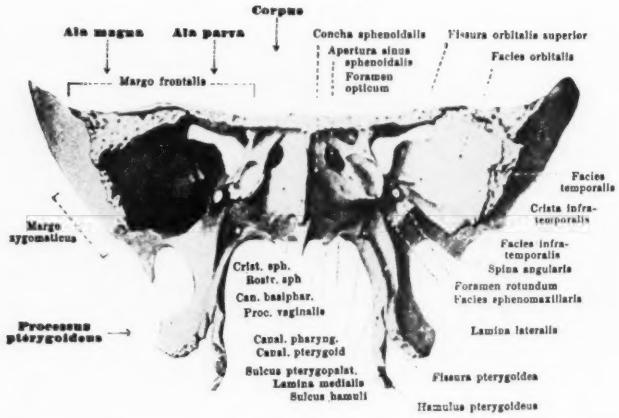

Figure 3 (Spalteholz). Anatomic structures near base of pterygoid process forming the posterior boundary of the pterygopalatine fossa.

Figure 4. Upper lip retracted: incision at gingivolabial junction: beginning elevation of periosteum.

Figure 5. Periosteum and skin retracted: antral wall opened: injection of sphenopalatine ganglion.

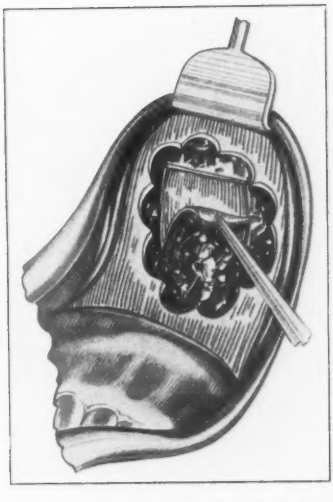

Figure 6. Lining mucous membrane dissected from posterior wall of antrum.

Figure 7. Breaking down posterior wall of the antrum with chisel.

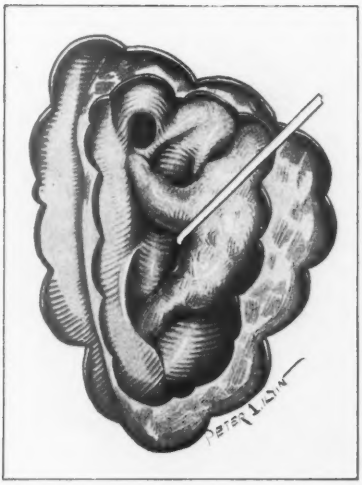

Figure 8. Sphenopalatine ganglion is contained in this mass of tissue, which has been partly dissected from its bed.

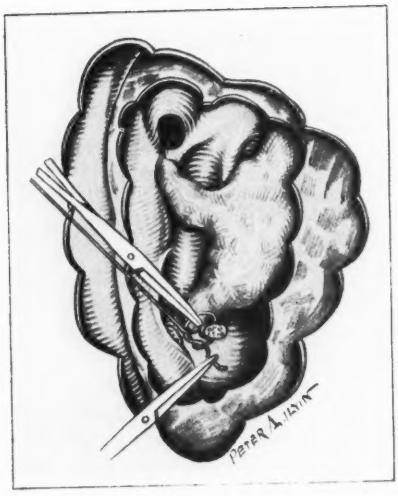

Figure 9. Cutting and ligation nn. palatini and a. palatina decendens.

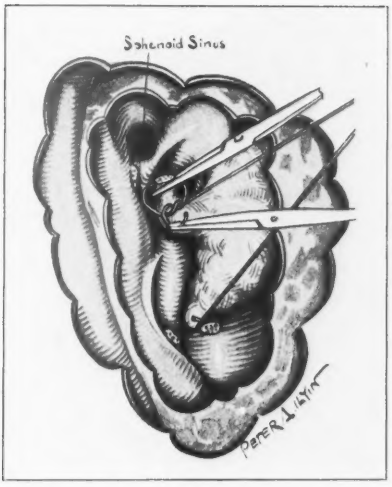

Figure 10. Cutting and ligating nn. Rami nasales posteriores superiores and a. sphenopalatina.

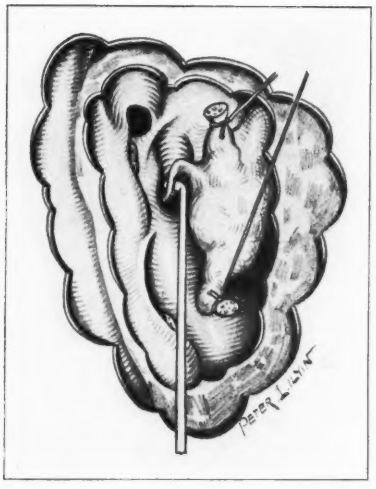

Figure 11. The mass containing the ganglion has been pulled aside: the nerve and artery canalis pterygoidei are freed for ligation and are shown lying on the hooked dissector.

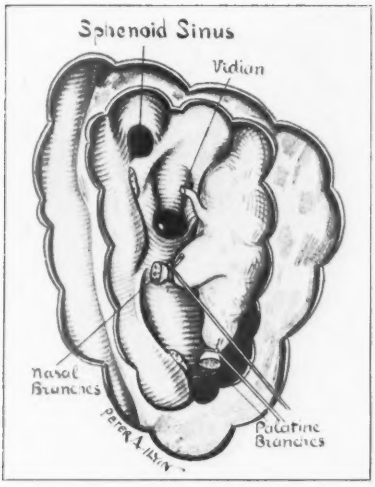

Figure 12. The nerve and artery canalis pterygoidei are shown severed: palatine and nasal branches ligated.

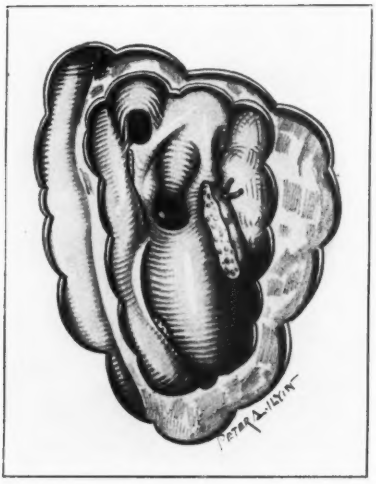

Figure 13. Ligature has been thrown about base of mass external to the sphenopalatine ganglion. Sphenopalatine ganglion has been removed.

II.

ANATOMIC AND SURGICAL CONSIDERATION OF CERTAIN CERVICAL ABSCESSES COMPLICATING MASTOIDITIS. REPORT OF CASES.*

By I. D. Kelley, Jr., B. S., M. D.,

St. Louis.

Cervical abscesses complicating mastoiditis are often puzzling to the aural surgeon when they are deep seated in the neck, and especially so when they are found remote from the primary infection in the mastoid process. An anatomic study of the mastoid and its adjacent structures clarifies greatly the "modus operandi" of these sinking neck abscesses and points to the surgeon the most efficient avenues for proper drainage and undelayed healing.

Illustration No. 1 represents the base of the temporal bone. At 1 is seen the mastoid process and to its mesial side the (2) incisura mastoideæ or digastric groove, while still farther inward is the groove for the (3) occipital artery. Above the digastric groove is the (4) stylomastoid foramen, while to the inner side of the stylomastoid process is the (5) jugular surface, and (6) jugular fossa, beyond which is found at (7) the carotid canal.

Illustration No. 2 is a view of the base of the skull, one-half of which pictures only the bony anatomy, while the other half shows its muscle attachments as well. At 1 again is seen the mastoid process and mesially to it (2) the digastric groove. On the opposite side, attached most externally to the mastoid process, is the (3) sternomastoid muscle. More internally and next in order is the (4) splenius capitis muscle, while still more deeply placed is the (5) longissimus capitis muscle, and finally the (6) digastric muscle is seen at its attachment along the digastric groove. Because of the attachment of these mus-

*Read before the Middle Section meeting of the American Laryngological, Rhinological and Otological Society, February 23, 1925, at Lafayette, Ind.

cles with their investing fascial sheaths to an infected mastoid process it will be necessary to trace their course through the neck. However, before doing so consideration must be given to the fascia colli or deep cervical fascia from which these muscle sheaths are derived. The deep cervical fascia of the neck presents many remarkable characteristics. It invests not only the muscles but forms aponeurotic coverings for the pharynx, trachea, esophagus, the glands and large vessels. Enclosing the sternomastoid muscle, it can be traced backwards over the trapezius and the deep muscles which it surrounds, finally blending posteriorly with the ligamentum nuchæ. It can be traced forwards to the midline of the neck, over which it forms a continuous membrane; a layer of this fascia enclosing the infrahyoid muscles passes across the midline of the neck in front of the trachea, and a third layer passes inward in front of the trachea, enclosing the thyroid body. Beneath the sternomastoid muscle the fascia helps to form the carotid sheath, which is completed in its formation by septal processes stretching inward across the neck in relation to the infrahyoid muscles, trachea, esophagus, pharynx and prevertebral muscles. Not only are the esophagus and trachea encapsulated in cervical fascia, but a septal layer passes across the midline of the neck between the two. Lastly a strong prevertebral fascia passes across the neck in front of the prevertebral muscles and behind the pharynx and esophagus.

Illustration No. 3 is a schematic representation of the general envelope of the deep cervical fascia, showing the processes and partitions which proceed from its deep surface, subdividing the neck into compartments capable of limiting and determining the spread of pus. At 1 is seen the most important central or visceral compartment. Here there is no barrier interposed between this compartment and the superior mediastinal space of the thoracic cavity. In front of this visceral compartment is a small (2) muscular compartment containing the depressor muscles of the hyoid bone, while on the inner surface of the sternomastoid muscle is found the important (3) vascular compartment, surrounded by the carotid sheath in which both the pretracheal and prevertebral septa take part in forming. The (4) prevertebral fascia stretching across the neck anterior to the prevertebral muscles is seen separating the

large posterior muscular or vertebral compartment from the visceral compartment.

Returning again to the consideration of the group of muscles inserted into the mastoid process, with their investing sheaths derived from the deep cervical fascia at its attachment along the mastoid process, it will be found that the course of each muscle as it proceeds to its distal attachment is different. The sternomastoid muscle descends downwards and forwards to the upper border of the sternum and is the most superficially placed muscle of this group in the neck. The splenius capitis and longissimus capitis muscles pass backwards and downwards under cover of the sternomastoid and trapezius muscles, the former having its insertion into the seventh cervical and six upper dorsal vertebræ, while the latter, under cover of the splenius capitis muscle, is inserted into the three or four upper dorsal vertebræ. The digastric muscle passes downwards, forwards and inward from the digastric groove to the intermediate tendon on the hyoid bone and thence upwards to the final attachment on the inner side of the lower jaw at the symphysis.

A serial study of cross sections through the neck show clearly the relation of these muscles one to the other. In a section taken just at the tip of the mastoid process the sternomastoid muscle will be seen to occupy a position most externally. Internal to the sternomastoid muscle and extending more posteriorly is the splenius capitis muscle. Deeper is seen the longissimus capitis muscle still attached to the mastoid tip, while still more internally is the digastric muscle in its relation to the rectus capitis lateralis muscle, carotid sheath and prevertebral fascia.

A cross section at a lower level shows the splenius capitis and longissimus capitis muscles assuming a more posterior position, and the digastric muscle is seen progressing forwards and now abuts the carotid sheath.

At the next lower level the splenius capitis and longissimus capitis muscle are still seen partially under cover of the sternomastoid muscle, but coursing farther backwards. The digastric muscle is now well anterior to the carotid sheath and on a line with the prevertebral fascia.

Finally, at a lower level the splenius and longissimus capitis muscles are occupying their most posterior position, while the digastric muscle is far anterior to the carotid sheath and prevertebral fascia, and is now found at the side of the visceral compartments of the deep cervical fascia. In the next cross section the digastric muscle by ascending has passed out of the picture.

It is now readily understood what an important bearing the course of these muscles, with their fascial sheaths in relation to the facial septa and compartments, have on the direction and progress of sinking abscesses of the neck complicating mastoiditis. It is so striking that Alexander devised the schematic arrangement in Illustration No. 4, showing into which muscle or fascial sheath a collection of pus will burrow as it leaves the mastoid through fistulæ on various aspects of its cortex. At 1 is seen an abscess in a cellular mastoid process, 2 represents a thickened external cortex. The abscess is hindered because of a thickened external wall from breaking outwards, so must perforate in an inward direction, thus forming a sinking abscess in (arrow No. 1) the sternomastoid splenius or longissimus capitis muscles or (arrow No. 2) into the fascia internal to these muscles, or still more mesially into the (arrow No. 3) digastric muscle itself. Pus infiltrating into the fascial sheath burrows more quickly than pus entering into muscle tissue, consequently in a remarkably short time it can reach in a forward direction the pleural apex and clavicle; behind it may reach as far as the scapulæ and neck vertebræ. It may form broad abscesses between the superficial and deep throat and neck muscles or abscesses in the neighborhood of the pharynx, which may spontaneously perforate into the pharynx itself. Especially is this true when the pus infiltration from the mastoid is into the digastric muscle or its immediate surroundings. Still further removed, the pus may even cross the midline of the neck, forming abscesses in opposite muscle groups.

Illustration No. 5 is taken from a normal mastoid process, showing a uniform thickness of the outer cortex. The tip is paper thin, while the inner wall is sufficiently thick to preclude the possibility of a perforation in the region of the digastric

groove. The contrast in Illustration No. 6 is striking. Here the pneumatic mastoid with its diploetic tip has an outer cortex quite thick, while the tip and inner cortex is extremely thin, predisposing to the formation of a fistulous opening on the mesial surface of the mastoid tip in the region of the digastric groove, as shown in Illustration No. 7, which pictures the bone pathology of a Bezold's mastoiditis with a sequestrum in the fistulous opening on the mesial side of the mastoid process. Because this pathologic condition was first described clinically by Bezold, sinking neck abscesses complicating mastoiditis with fistulæ in and about the digastric groove are called "Bezold's mastoiditis." Bezold has shown, by much careful and painstaking anatomic and experimental investigation, that not only borings through the mastoid cortex at designated points perforate into certain muscles, but that injections of a colored gelatin mass into these borings follow well defined pathways, permitting its recovery from definite muscle groups. This makes it now necessary to consider the pneumatic and diploetic surface of the inner cortex of the mastoid process. Illustration No. 8 represents a simple mastoid operation. At 1 is seen the inner cortex covering the lateral sinus; 2 is the mastoid antrum; 3 the horizontal semicircular canal, while externally and below this, extending as far down as the mastoid tip between the lateral sinus on the one side and the posterior canal wall containing the facial nerve on the other side, is a (4) deep groove fashioned from a thick diploetic bony structure found to a greater or less degree in all mastoids. It is here at 5, a point representing the apex of a funnel surrounded above by the horizontal semicircular canal, behind by the lateral sinus, in front by the facial nerve, and below by the bulb of the jugular vein, where perforations in the region of the digastric groove occur. When pus during a mastoid operation is seen to well from a fistula in this location, not only is the surgeon faced with the knowledge that to proceed further in this operative field is impossible because of injury to the important surrounding structures, but he must now decide where and how a sinking abscess of the neck can best be drained. A careful examination of the location of the inner cortex fistula in its relation to the digastric groove and attached tip muscles with their facial sheaths, together with the

previous consideration of all symptoms suggesting a cervical abscess complicating the mastoiditis, should do much to solve the problem of attack. Especially when it is borne in mind that fistulæ found perforating the mastoid tip more externally are prone to simple sternomastoid muscle infiltration. Those found more externally on the inner cortex dispose, because of the attachment and backward curve of the splenius and longissimus capitis muscles, to postoccipital or postvertebral abscesses, while deeply located fistulæ, found more mesially on the inner cortex, perforate the digastric groove, permitting the pus to follow the digastric muscle and becoming eventually a prevertebral or retropharyngeal abscess. This general scheme is subject to modification in the individual case, because it must be borne in mind that the vessels and nerves in this region, some of them piercing the deep cervical fascia, must influence the direction in which the pus will burrow. As example, the occipital artery coursing backwards from the occipital groove finally piercing the trapezius muscle near the external occipital protuberance to reach the scalp; the course of the spinal accessory nerve and the hypoglossal nerve as it passes internal to the mastoid process in an anterior direction to the tongue offer avenues of escape for pus into other facial compartments.

The experience of many observers has shown that Bezold abscesses drain slowly and unsatisfactorily and that surgical drainage must be performed with a wide incision reaching, if possible, the lowest point of the abscess, permitting free open drainage. The mastoid operative field is only available in cases of simple pus infiltration into the upper fibers of the sternomastoid muscle, consequently the neck then becomes of necessity the operative field of choice.

Illustration No. 9 represents the cervical triangles of the neck; 1 the anterior triangle anterior to the sternomastoid muscle separated into its three subtriangles by the digastric muscle above and the omohyoid muscle below; 2 the posterior triangle behind the sternomastoid muscle, divided by the omohyoid muscle into its upper and lower subdivisions. If the surgeon in draining these abscesses will carry his incision from below the mastoid tip well down towards the clavicle in the posterior triangle an inward dissection will then bring him

within easy reach of the splenius and longissimus capitis and other allied postvertebral muscle groups. A dissection inward but more anteriorly will easily reach the carotid sheath and digastric muscle and, if continued inwardly behind the prevertebral fascia, the surgeon will finally reach the longus colli muscles on the anterior body surface of the cervical vertebræ. Drainage of a cervical abscess from such an incision is not only surgical but prompt, because the most dependent portion of the abscess can be reached, and because the posterior triangle affords sufficient length and depth for such an incision and dissection. It will prove in many cases more successful than an attempted drainage about the mastoid process, under the occiput posteriorly, in the anterior triangle or through the posterior pharyngeal wall, as is so frequently attempted.

I desire to give very briefly three case reports and but mention a fourth case recently come to my attention.

Case 1.—Adult, male. History: Three months previously, complicating "influenza," the patient had an otitis media suppurativa, treated locally by his physician. Ear discharge continued with but only slight improvement. Three weeks before consulting me a severe head cold caused an exacerbation of the ear symptoms, followed by deep pain and stiffness in the neck muscles on the affected side. Examination: Right ear showed a sagging posterior superior canal wall, a small posterior quadrant perforation in a thickened subacutely inflamed drum membrane, slight creamy purulent discharge from the perforation. No postauricular infiltration or tenderness was noticeable, but there was marked tenderness below and behind the mastoid tip, with restricted head movements due to rigidity of the sternomastoid and trapezius muscles; no visible swelling was noticed in this region. Temperature 100°. Referred to St. Mary's Infirmary for a simple mastoid operation. In hospital X-ray showed only a suspicious mastoid involvement. Pus culture report from the ear was streptococcus hemolyticus. At operation the mastoid cortex was normal and thick. Pneumatic cells revealed little or no destruction. Antrum contained pus, and diploetic tissue from antrum to tip along posterior canal wall contained mucopus, and, when finally removed, pus welled from a fistula at a deep point surrounded by the posterior wall, the horizontal semicircular canal, lateral sinus

and the bulb of the jugular vein. The significance of this finding was not fully appreciated, and the hope was entertained that adequate drainage had been established. The mastoid wound was dressed and the patient returned to bed. At subsequent dressings pus welled freely from the inner cortex fistula on external pressure over the sternomastoid and trapezius. The operative field remained lusterless, showing no disposition to granulate. The mastoid pus cultured was streptococcus hemolyticus; blood culture was negative. Daily temperature reached approximately 100 and 101 degrees. A second operation was advised and done. A posterior neck incision was made over the anterior border of the trapezius muscle, and the dissection carried upward and forward well under the mastoid tip until the mesial tip surface was reached, with the evacuation of only a small quantity of pus. Wound dressing continued. Pus continued welling from mastoid fistula on pressure. The neck was stiff, and eventually the patient complained of a sore throat and difficulty in swallowing. No retropharyngeal abscess was noted, but the posterior pharyngeal wall looked red and possibly indurated. A third operation was advised and done. A wide vertical incision was now made in the posterior cervical triangle, with a dissection carried inward posterior to the carotid sheath until the anterior body surfaces of the cervical vertebræ were reached. A large quantity of pus was then evacuated, and on digital exploration of the neck wound both the transverse spinous processes and bodies of the vertebræ could easily be felt. Drainage was now satisfactory. The mastoid wound healed, and at the end of the sixth hospital week, because of the patient's hemolyticus infection and marked malnutrition, he was advised to return home where superficial neck wound dressings could be continued in fresher and happier surroundings.

Case No. 2.—Adult, male. Entered St. Luke's Hospital from my office for a simple mastoid operation. Examination revealed a subacute otitis media suppurativa and a large postauricular subperiosteal abscess with induration extending into the neck. The patient stated that the swelling behind the ear had existed for approximately four weeks. At operation a large external cortex fistula was found. The mastoid cells were full of pus, necrotic and destroyed. The mastoid tip, because

of necrosis, was removed, and pus infiltration into the fibers of the mastoid muscle was noted. Postoperative healing was slow, with pus welling from the region of the mastoid tip. Patient complained of stiff neck. Pressure over the neck muscles on the affected side caused a flow of pus from the lower portion of the mastoid wound. On his discharge from the hospital patient came to my office for daily dressings. The pus discharge and sore stiff neck continued until he entered St. John's Hospital for a second operation, where I made a posterior triangle incision and deep dissection, starting from the lower end of the mastoid incision. This drained an abscess posterior and internal to the carotid sheath. Shortly after and during my absence from the city the neck wound was permitted to close from below upwards until when he was seen by me again pus was once more flowing from a small opening in the region of the mastoid tip. He again complained of a stiff sore neck, followed rapidly by pain in his throat and difficulty in swallowing. Pharyngeal examination showed an indurated and slightly swollen posterior pharyngeal wall. Under local anesthesia an incision was made through this wall, but no pus was evacuated. Eventually, after an absence of several days from my office, the patient returned, imparting the information that following an attack of severe pain in the upper back part of his throat and an unusual difficulty in swallowing he had spit up a quantity of pus, followed by an immediate relief from all symptoms. This incident was followed by a speedy closure and complete healing of his mastoid wound.

Case No. 3.—Adult, female. Entered Barnes Hospital from Washington University Dispensary with an acute mastoiditis complicating a subacute purulent otitis media. At operation I found a beginning subperiosteal abscess, a small fistula through the outer mastoid cortex, pus and bone necrosis extending to and including the mastoid tip. On removing the mastoid tip a small abscess was evacuated from the mastoid muscle fibers. Postoperative healing was slow, because of pus welling from the lower angle of the wound. The neck became sore, stiff and indurated. Again the patient entered the hospital from the clinic, where postoperative dressings were being continued, and a posterior triangle incision was carried by dissection into the fascia in the inner surface of the sternomastoid muscle.

where an abscess was again evacuated. Complete healing speedily followed.

Dr. H. W. Loeb and Dr. W. T. Coughlin have discussed with me a recent case of theirs where, following a simple mastoid operation performed by Dr. Loeb, Dr. Coughlin, through a posterior triangle neck incision and dissection on the affected side, evacuated a large sinking abscess. In a few days Dr. Coughlin was compelled to repeat this operation on the opposite side of the neck, because of a second pointing of pus in that location.

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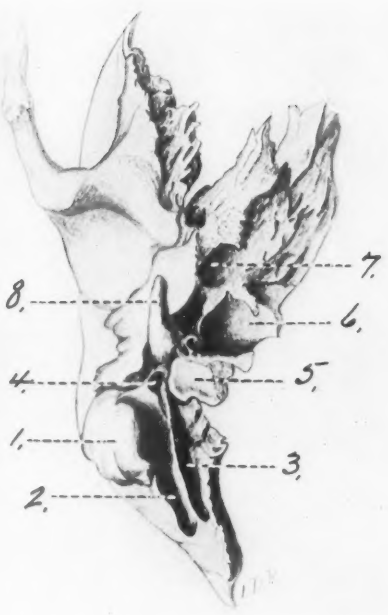

Illustration No. 1.

The base of the temporal bone. 1. Mastoid process. 2. Incisura mastoideæ (digastric groove). 3. Groove of the occipital artery. 4. Stylomastoid foramen. 5. Jugular surface. 6. Jugular fossa. 7. Carotid foramen. 8. Styloid process.

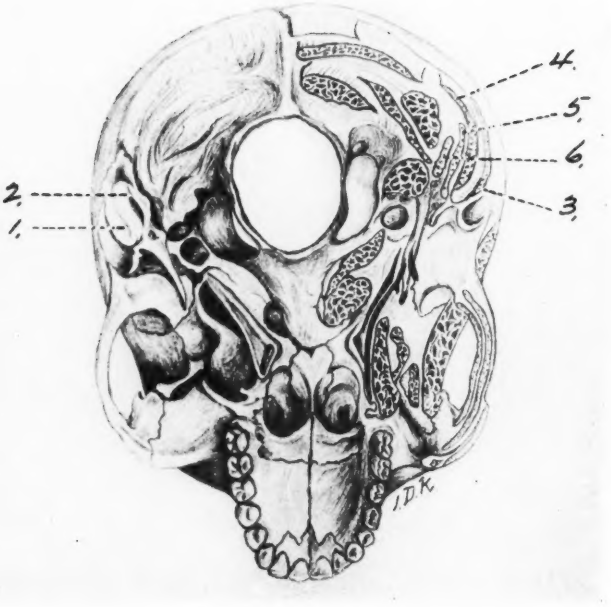

Illustration No. 2.

The base of the skull, one-half of which pictures only the bony anatomy, the other half showing its muscle attachments as well. 1. Mastoid process. 2. Digastric groove. 3. Sternomastoid muscle. 4. Splenius capitis muscle. 5. Longissimus capitis muscle. 6. Digastric muscle.

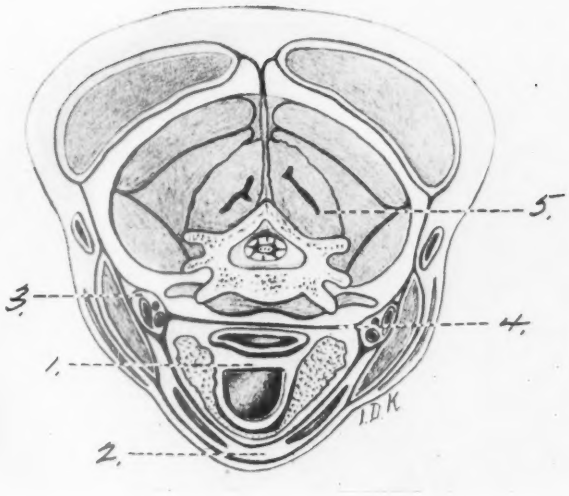

Illustration No. 3.

Schematic representation of the general envelope of the deep cervical fascia. 1. Central or visceral compartment. 2. Muscular compartment containing depressor muscles of the hyoid bone. 3. Vascular compartment. 4. Prevertebral facia. 5. Posterior muscular or vertebral compartment.

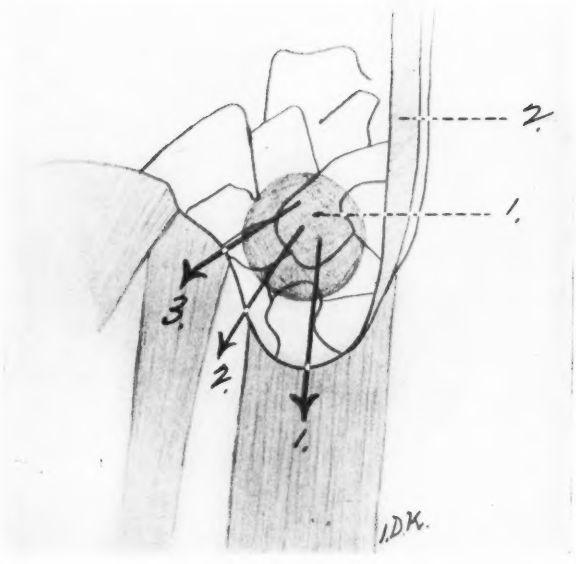

Illustration No. 4.

Schematic arrangement showing into which muscle or fascial sheath a collection of pus will burrow as it leaves the mastoid through fistulæ in various aspects of its cortex. 1. Abscess. 2. Thickened external cortex. Arrow 1. Pus burrowing into sternomastoid, splenius and longissimus capitis muscles. Arrow 2. Into fascia internal to sternomastoid splenius and longissimus capitis muscles. Arrow 3. Pus burrowing into digastric muscle.

Illustration No. 5.

Normal mastoid process showing a uniform thickness of the outer cortex, a tip paper thin, an inner wall sufficiently thick to preclude the possibility of a perforation in the region of the digastric groove.

Illustration No. 6.

Pneumatic mastoid with a diploetic tip, a thick outer cortex, an extremely thin tip, and inner cortex predisposing to the formation of a fistulous opening on the mesial surface of the tip in the region of the digastric groove.

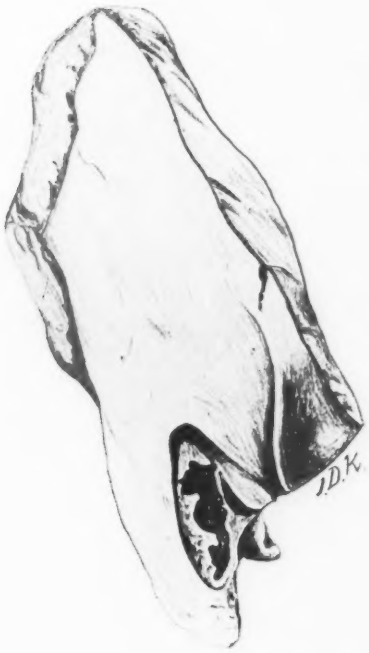

Illustration No. 7.

Bone pathology of a Bezold's mastoiditis with a sequestrum in the fistulous opening on the mesial side of the mastoid process.

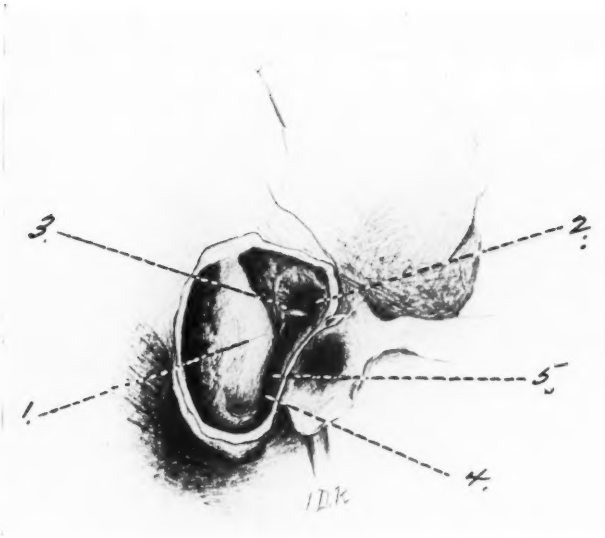

Illustration No. 8.

Simple mastoid operation. 1. Inner cortex covering the lateral sinus. 2. Mastoid antrum. 3. Horizontal semicircular canal. 4. Deep groove fashioned from a thick diploetic bony structure. 5. Point representing the apex of a funnel where perforations into the region of the digastric groove occur.

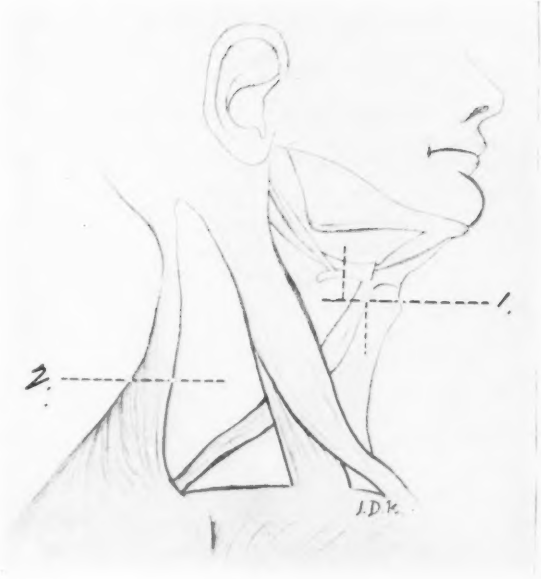

Illustration No. 9.

Cervical triangles of the neck. 1. Anterior triangle. 2. Posterior triangle.

III.

ARTIFICIAL PNEUMOTHORAX IN THE TREATMENT OF PULMONARY TUBERCULOSIS AND ITS EFFECTS ON THE LARYNX.*

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The purpose of this paper is to record my observations on artificial pneumothorax as employed in the treatment of pulmonary tuberculosis, with special reference to its effects on the larynx.

It is not my aim, however, to discuss artificial pneumothorax in all its details, but in order to show its influence on the larynx it will be necessary to review the subject briefly.

Artificial pneumothorax has been employed in the treatment of pulmonary tuberculosis since 1895. It was then introduced by Dr. Carlo Forlanini, of Pavia, Italy. In the United States it was first used by Dr. John B. Murphy of Chicago in 1898, and independently of Dr. Forlanini.

Until 1910, however, this therapeutic measure received only local attention. During the past fifteen years, artificial pneumothorax has been extensively applied in certain cases of pulmonary tuberculosis, and if these cases are properly selected, both the immediate and ultimate results are extremely gratifying.

The modus operandi of artificial pneumothorax is based on the principle of rest to the affected lung. This is obtained by insufflating the pleural space on the affected or more affected side with nitrogen gas or filtered atmospheric air, thus completely or partly compressing and immobilizing the lung. By so doing, the diseased areas of the lung are squeezed together, the walls of the cavities are brought into apposition, detritus and purulent matter are expressed, fibrosis is promoted and

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the lesion gradually allowed to cicatrize. While the lung is thus being reduced in size, the inhaled air, as well as the arterial blood supply and the lymph circulation become diminished, with a consequent decrease in physiologic activity. It can, therefore, be readily seen how all the factors concerned in the production of this changed pathology tend to increase fibrosis, and in that way bring about healing of the diseased parts.

The ideal case for treatment by artificial pneumothorax is one:

First—Where the lesion is confined to one lung, although it is often employed in cases where the contralateral lung is affected, but to a lesser extent.

Second—Where the lesion is steadily progressive and presents signs of softening and cavitation, as demonstrated by physical signs and X-ray examination.

Third—Where the pleura on the affected side is free, or nearly free, from adhesions.

Fourth—Where the symptoms, such as fever, cough, expectoration or hemoptysis are troublesome and do not yield to medicinal treatment.

Fifth—Where other well known accepted methods of treatment have been tried in a case, as described above, and failed.

Generally speaking, artificial pneumothorax is indicated in the progressive type of tuberculosis, mainly in the advanced or rapidly advancing cases, where a great deal of destruction of pulmonary tissue has taken place, particularly where the lesion is unilateral, where the symptoms are very troublesome, and where the hygienic dietetic method has been given a fair trial and has met with failure.

The selection of cases varies with different operators. A few are radical in their tendencies, and advocate the employment of artificial pneumothorax in incipient cases. The majority of men, however, are still conservative and employ artificial pneumothorax in the more advanced cases, and in the earlier cases only after the usual methods have proved unsuccessful.

In going over the records of my patients treated by artificial pneumothorax, I was impressed with the fact that not in a single instance where the treatment was successfully carried out had laryngeal tuberculosis occurred as a complication.

During the past two years, therefore, I have concentrated my attention on the effects of artificial pneumothorax on the larynx, and so far I have never observed laryngeal tuberculosis develop in the course of treatment, nor did I ever see this complication occur subsequently in a patient cured or arrested by means of artificial pneumothorax.

Upon further study, I also noticed that several of my patients who received artificial pneumothorax treatment for their pulmonary lesion, and who also presented a coexisting laryngeal lesion, showed definite improvement in the larynx. This was especially true in the subacute type of laryngeal tuberculosis. Under this adjunctive treatment I observed that infiltrations of the larynx were absorbed, granulations disappeared and ulcerations healed. This, of course, is in accordance with the general understanding that where the pulmonary lesion improves the laryngeal lesion improves also.

In a paper which I read before the New York Academy of Medicine in 1914,[1] and which was based on a study of 500 cases of pulmonary tuberculosis in adults, I stated that this complication was present in 25.6 per cent of all cases of pulmonary tuberculosis, 12 per cent being found among incipient cases, 26.4 per cent in moderately advanced cases and 45.3 per cent in far advanced cases. Statistics of other observers fairly agree with my findings.

Since all my patients treated by artificial pneumothorax presented very active and progressive lesions and were in the advanced stage of the disease, and in view of the fact that a great number of cases of this type sooner or later develop tuberculous lesions of the larynx, the complete absence of this complication in pneumothorax cases is a very striking fact and is worthy of attention.

As you know, so far there has been no specific remedy discovered for the cure of pulmonary tuberculosis, and the remedy which will some day be found to cure this disease will also heal the laryngeal lesion. Until such time, however, in addition to all accepted methods of treatment, we must also use all available means of prophylaxis, and, in my experience, artificial pneumothorax, successfully carried out in suitable cases, is a positive way to prevent this unfortunate complication. Furthermore, in cases where the laryngeal lesion is

already in existence, artificial pneumothorax exerts a beneficial influence on its course and often cures this complication entirely.

Before recording my observations, it occurred to me that it would be well to gather data from other men and compare their findings with my own. However, as nothing has ever been written on the subject, at least so far as I know, I resorted to the writing of personal communications to a number of men who have had extensive experience with artificial pneumothorax, and the following tables indicate the opinions of the various observers:

TABLE No. 1.

AUTHOR	Number of Cases Pulmonary Tuberculosis Treated by Artificial Pneumothorax	Number of Cases Developing Tuberculous Laryngitis
Blanchet, S. F.	200	1
Brown, Lawrason	100	2
Howk, H. J.	37	0
Kinghorn, H. M.	100	0
Landis, H. R. M.	200	0
Peters, Andrew	91	0
Peters, LeRoy	600	0
Parfitt, C. D.	150	1
Stivelman, B.	114	0
Total	1592	4

TABLE No. 2.

AUTHOR	Number of Cases Laryngopulmonary Tuberculosis Treated by Pneumothorax	Number of Cases Improved	Number of Cases Unimproved	Number of Cases Stationary
Blanchet, S. F.	5	4	1 died	
Dworetzky, J. P.	6	5	1 died	
Goodall, H. S.	2	2		
Kinghorn, H. M.	1	1		
Parfitt, C. D.	2	2		
Peters, A.	16	12		4
Total	32	26	2	4

From Table No. 1 it is readily seen that out of 1,592 cases of pulmonary tuberculosis treated by artificial pneumothorax only four developed tuberculous laryngitis. Of these, two were reported by Dr. Lawrason Brown, and, according to his statement, it is quite probable that the laryngeal lesion existed previous to the time artificial pneumothorax was administered.

From Table No. 2 we learn that out of thirty-two cases

of laryngopulmonary tuberculosis treated by artificial pneumothorax, twenty-six cases were either cured or showed definite improvement in the laryngeal lesion. Four remained stationary and two died. The four stationary cases, according to the report of the Loomis Sanatorium, did not have a satisfactory collapse of the actively secreting cavities.

The prophylactic, as well as the remedial, action of artificial pneumothorax on laryngeal tuberculosis is best explained by the beneficial influence exerted on the pulmonary lesion. As we all know, tuberculosis of the larynx is always secondary to tuberculosis of the lungs. By compressing the affected lung we eliminate the original source of infection, and thus spare the larynx from the constant bathing by bacilli laden sputum. Furthermore, by relieving the cough, we do away with all unnecessary trauma of the larynx and thus prevent the formation of a locus minoris resistentiæ.

The following are abstracts of the letters of the various observers, giving their opinion on the effects of artificial pneumothorax on laryngeal tuberculosis.

Dr. B. Stivelman, New York, January 13 and 17, 1925: My records show that in the past seven and one-half years 114 patients received pneumothorax therapy for a considerable length of time, with at least partial benefit to 81 patients. A perusal of my records does not disclose a single instance of the development of an active tuberculous laryngitis in a patient successfully treated by artificial pneumothorax. I have had several patients in whom a preexisting mildly active laryngeal involvement was greatly benefited simultaneously with the improvement in the lung condition after the induction of pneumothorax. As a matter of fact, I am beginning to realize that tuberculous laryngitis, provided the epiglottis is not involved, is no contraindication to this mode of therapy.

Dr. LeRoy S. Peters, Albuquerque, N. M., January 30 and March 6, 1925: In my thirteen years' experience with artificial pneumothorax, with about 600 cases, I have never seen a case develop laryngeal tuberculosis during the course of treatment, and I have never seen a patient with a tuberculous larynx on whom a pneumothorax was performed get worse during the treatment. Many of these cases have improved, and

the larynx became healed coincident with the healing of the pulmonary lesion. I am convinced that a tuberculous larynx is not a contraindication for pneumothorax treatment.

Dr. Lawrason Brown, Saranac Lake, N. Y., February 2 and March 9, 1925: There are only two out of 100 cases that Dr. Hayes, who does most of my gas work, can recall in which tuberculous laryngitis developed after collapse. Neither of these had throat examinations previously, and it may be that the condition had been there.

Dr. Andrew Peters, Loomis Sanatorium, January 21, 1925: Out of 107 patients who might be said to have a more or less satisfactory pneumothorax, sixteen are recorded as having laryngeal lesions at the time when pneumothorax was begun and none are recorded as having developed during the course of treatment. Of these sixteen patients with complicating laryngeal lesions, twelve showed definite improvement in the laryngeal lesion under treatment, and in four the lesions remained practically stationary. However, it might be noted that none of these four had a complete collapse of actively secreting cavities.

Dr. H. R. M. Landis, Philadelphia, February 10 and March 5, 1925: As far as my observation goes, I have never seen laryngeal lesion develop in the course of pneumothorax treatment except in those cases that were in a serious condition and in which pneumothorax had been given as a last resort. This is based on experience with about 200 cases.

Dr. Hugh M. Kinghorn, Saranac Lake, N. Y., February 13, 1925: I have had upwards of 100 cases of artificial pneumothorax and have had no cases of pulmonary tuberculosis so treated develop laryngeal lesions. I had one case who had definite laryngeal involvement, in which the larynx showed great improvement under pneumothorax treatment.

Dr. Sidney F. Blanchet, Saranac Lake, N. Y., February 21, 1925: I can report only one frankly developed case of tuberculous laryngitis in 200 cases treated by pneumothorax. I can find only one case developing laryngeal lesion during the course of successful treatment by artificial pneumothorax. It is a case that has been under treatment about one year. The lesion is a mild one with slight edema and erythema of the cords and arytenoids but no ulceration. Because the patient

is doing well in every other way, I believe the larynx will improve. In regard to changes in laryngeal lesions which were present at the time the artificial pneumothorax was started, I have found only five frankly developed cases of laryngeal tuberculosis. These cases, as you may imagine, were not among those most favorable for treatment. In four of them, however, there has been definite improvement. One is dead. Careful investigation would probably have shown a goodly number who had slight laryngeal lesion at the time artificial pneumothorax was started, and probably a favorable response to pneumothorax treatment prevented the development of the lesion or led to its clearing up. It would seem to me unfair to artificial pneumothorax to quote these figures unless the condition of the larynx had been carefully investigated before and after treatment, because if improvement follows in advanced laryngeal cases it is reasonable to believe that it is much more helpful in cases with a slight early laryngeal lesion.

Dr. C. D. Parfitt, Calydor Sanatorium, Gravenhurst, Ont., February 11, 1925: I should think I have altogether done in the last twelve years about 150 pneumothoraces. In only one case have I recollection of the development of laryngeal lesion in the course of treatment. This case is comparatively recent and the laryngeal lesion has subsided, but pneumothorax was done at the height of an acute relapse and the laryngeal involvement followed shortly after. Of cases having a laryngeal lesion before treatment was begun, one patient, about 1915, presented himself with a severe involvement at the base of the epiglottis and left ventricular band. This was treated by galvanocauterization and by artificial pneumothorax. Both measures, I think, contributed to its arrest. This patient did surprisingly well and the laryngeal lesion healed entirely, and, so far as I know, gave no further trouble and the patient is still living. Another patient, admitted with very acute process and with an eruption of miliary tuberculosis on the left ventricular band, under silence and pneumothorax made a surprising recovery. The laryngeal lesion cleared up entirely. A third case had a doubtful interarytenoid infiltration when pneumothorax was induced. I cannot say definitely that it was tuberculous. It may have been due to cough and sputum, with an associated pachydermatosis, but it quite subsided. Another

somewhat similar case subsided under treatment. My impression is that artificial pneumothorax is an admirable adjunct to the treatment of laryngeal lesions.

Dr. J. Burns Amberson, Jr., Loomis Sanatorium, January 28, 1925: I do not recall having seen a case in which this complication developed so long as the lung disease was definitely benefited and brought to quiescence. This is in conformity with our usual experience that laryngeal lesions, if present, parallel the changes in the pulmonary lesion, whatever it may be. It is unusual for us to see laryngitis develop while the pulmonary lesion is quiescent or healing.

Dr. Nathan Barlow, U. S. Veterans' Bureau, January 28, 1925: I have seen laryngeal lesions develop in cases in whom artificial pneumothorax has been induced, but in such cases I do not consider that the treatment has been successful.

Dr. H. Schwatt, New York, February 14, 1925: I find no record of any cases who have been successfully treated by artificial pneumothorax developing laryngeal complications in the course of treatment.

Dr. H. S. Goodall[2] reports two cases of laryngeal tuberculosis with ulcerations of larynx and granular masses in the posterior commissure, both cases having cleared up under pneumothorax treatment.

METHOD OF TREATMENT.

The administration of artificial pneumothorax, although simple in itself, like many other operations, requires considerable skill. The operator should not only be fully trained in its technic, but should also be thoroughly experienced in the diagnosis and treatment of tuberculosis.

The necessary apparatus for pneumothorax work may be a very simple affair, consisting of two graduated bottles connected by rubber and glass tubing, with a few stopcocks. I prefer to use Dr. Samuel Robinson's apparatus (Fig. A) on account of its simplicity and convenience. This consists of two graduated 2,000 c. c. bottles. Bottle "A," which may be raised during the operation, contains sterile water or some antiseptic solution, and bottle "B" contains gas or atmospheric air. Bottle "B" is connected through a T-tube with a water

manometer and also with a rubber tube, to which an appropriate needle is attached.

The chest should be carefully studied in order to determine the site for the puncture. We should try to select an area which is free from adhesions and, in addition to physical examination, should resort to the X-ray and fluoroscope. Occasionally, however, adhesions are encountered in spite of utmost precautions taken, and a trial puncture is the only positive method available. The usual site for the puncture is in the axillary region, either in the anterior or posterior lines, in the fourth, fifth or sixth intercostal spaces. The patient lies on the untreated side (Fig. B), and the site of the operation is prepared in the usual surgical manner. Strict aseptic precautions should be taken. The skin is anesthetized with 1 per cent procain or novocain-adrenalin solution by means of a 1 cc. syringe, with a needle about 1½ inches long, and of No. 20 gauge. Personally, I prefer to use the same needle for both the anesthetic and the insufflation. After anesthetizing the skin, the needle is slowly inserted through the subcutaneous tissues, muscles and fascia, and just before reaching the parietal pleura a few drops of the solution are again injected. After waiting thirty to sixty seconds the needle is pushed through the parietal pleura, the syringe is removed, leaving the needle in situ, and the manometer is then turned on. If the point of the needle is in the pleural space the manometer fluid is sucked up, as shown by the negative pressure in the manometer. Patient is then told to breathe normally and the oscillations of the manometric fluid are observed. We are then certain that we are in the pleural space. The manometer is then turn off and the gas allowed to flow in.

Manometric readings should be taken after each 50 to 100 c. c. of gas is insufflated. The amount of gas to be used at each insufflation varies with the individual patient, usually from 200 to 400 c. c. at one time. As the gas is injected into the pleural space, the intrapleural pressure becomes less and less negative, and after a sufficient amount of gas is introduced it will turn positive. During the operation the manometric pressure serves as a fair index as to the amount of compression being attained, and also as to the presence or absence of adhesions or fluid.

The operation is at first repeated in one or two days, and later as often as may be indicated. The patient should be fluoroscoped both before and after each insufflation and X-rayed after each fourth or fifth treatment. Ambulant patients should be told to rest for a period of at least twenty-four hours after each treatment.

There are certain complications that may be encountered in the course of treatment:

1. Subcutaneous emphysema, which consists of infiltration of air or gas into the subcutaneous tissues of the chest wall near the site of the puncture. This, however, is of no serious consequence and can be avoided by using a needle of finer caliber.

2. Pleural effusion, which occurs in about 30 to 50 per cent of the cases some time in the course of treatment. This, as a rule, is not harmful in itself, except that it often tends to obliterate the pleural cavity, and pneumothorax treatment may have to be discontinued sooner than is desirable.

3. Pyothorax.—This occurs in a very small number of pleural effusion cases where the fluid becomes infected.

4. Spontaneous Pneumothorax.—This may occur as a complication due to a tear of the visceral pleura.

5. Pleural Shock.—A very rare occurrence and can be avoided by thorough anesthetization of the pleura before injecting the gas.

6. Gas or Air Embolism.—Extremely serious, but very rare. It may happen in case air enters into one of the pulmonary veins.

Fortunately, all the above complications, with the exception of pleural effusion, are of rarest occurrence and should not prejudice us against the use of artificial pneumothorax in properly selected cases.

SUMMARY.

Patients should be individualized and carefully observed before artificial pneumothorax is begun. It should be remembered that, at best, artificial pneumothorax is only an adjunct in the treatment of pulmonary tuberculosis, and the general hygienic-dietetic treatment should be employed for a period of at least six to eight months, even though the patient may be perfectly free from symptoms.

CONCLUSIONS.

1. Artificial pneumothorax, successfully carried out in properly selected cases, will prevent the development of tuberculous laryngitis in more than 99½ per cent of cases of active pulmonary tuberculosis thus treated.

2. Artificial pneumothorax is an extremely valuable measure in the treatment of suitable cases of laryngopulmonary tuberculosis, and when applied promptly and efficiently, improvement must follow in the larynx as well as in the lungs.

Case No. 1.—Advanced pulmonary tuberculosis treated by artificial pneumothorax, with complete success:

H. B., age 21, came under my care November 3, 1922. Diagnosis: Pulmonary tuberculosis, far advanced and active. Physical findings: Widespread infiltration of the left lung, with cavity formation in left upper lobe, with a great deal of softening. See Plate 1, Fig. 1.

Symptoms: Troublesome cough, with profuse purulent expectoration, pronounced tachycardia. Pulse range 130 to 150, at rest. Routine hygienic-dietetic treatment over a period of six months had no effect. Artificial pneumothorax was begun June, 1923, with immediate improvement. After three months of treatment all symptoms disappeared. Cough and expectoration ceased; there was no dyspnea; pulse ranged from 80 to 90. See Plate 1, Fig. 2.

He was then allowed to return to his home in New York but to continue with pneumothorax treatment. He soon returned to work as a taxi driver in New York. Examination of February 17, 1925, twenty months after treatment was begun, showed that the left lung is still well compressed. Patient is completely free from symptoms and is still working as taxi driver. Leads a quite normal life. Larynx is normal.

Case No. 2.—Acute laryngeal tuberculosis cleared up by artificial pneumothorax:

M. T., age 36. History of tuberculosis for about fifteen years. Diagnosis: Pulmonary tuberculosis, far advanced and active. Tuberculous laryngitis of acute type. Physical examination of chest revealed extensive infiltration of left lung with cavity formation in left lower lobe. See Plate 2, Fig. 1.

Examination of larynx showed pear-shaped infiltration of both arytenoids, swelling of posterior commissure, with edema of both vocal cords. See Plate 3, Fig. 1.

Symptoms: Fever 102 to 103 every afternoon for three months; troublesome cough with purulent expectoration; pronounced hoarseness and dysphagia. To all appearance, patient was in a terminal stage.

Pneumothorax was induced in left chest, resulting in partial compression, complete compression being prevented by numerous adhesions. See Plate 2, Fig. 2.

There was pronounced symptomatic improvement. Cough and expectoration were much diminished; temperature became normal. Symptoms referable to the larynx disappeared with the other symptoms, and examination of the larynx four months later showed all structures of the larynx apparently normal. See Plate 3, Fig. 2.

Case No. 3.—Advanced laryngopulmonary tuberculosis treated by artificial pneumothorax, complicated by spontaneous pneumothorax with effusion, followed by improvement:

S. G., age 28. History of tuberculosis for six years. Diagnosis: Pulmonary tuberculosis, far advanced and active. Tuberculosis of larynx, subacute type. Physical findings consisted of widespread infiltration of the left lung, with large cavity formation in left upper lobe, also some infiltrative changes in right lower lobe. See Plate 4, Fig. 1.

The larynx presented diffuse infiltration of both vocal cords with considerable thickening at posterior commissure. See Plate 5, Fig. 1.

Patient complained of troublesome cough and very copious purulent expectoration, of about sixteen ounces in twenty-four hours, with febrile exacerbations in the afternoon. Patient was very hoarse and at times aphonic. Treatment: After fifteen months' hygienic-dietetic treatment, it was decided to compress the left lung. A few insufflations were given in October and November, 1923, when patient developed a spontaneous pneumothorax, followed in a few days by a large pleural effusion. This was accompanied by high fever. Patient was kept in bed and fever gradually subsided, and it was decided not to aspirate. In the course of three or four months the fluid was apparently absorbed. See Plate 4, Fig. 2.

At present patient is enjoying good health. Cough is very slight; expectoration is very small in amount, about one-fourth of an ounce during the day as compared with sixteen ounces previous to collapse. The larynx shows pronounced fibrosis of both vocal cords, with permanent thickening at the posterior commissure. All other structures are perfectly free. See Plate 5, Fig. 2. Patient has a good voice of rather low pitch.

Case No. 4.—Advanced laryngopulmonary tuberculosis treated by artificial pneumothorax definitely improved:

M. V., age 28. Diagnosis: Pulmonary tuberculosis, far advanced and active. History of tuberculosis for about six years. X-ray findings of November 9, 1922, showed heavy infiltration of right upper lobe, with evidence of cavitation to a moderate extent. Slight infiltration of middle lobe, merging into peribronchial deposits at base. Left lung apparently clear. See Plate 6, Fig. 1.

The larynx showed a large infiltrative mass in the posterior commissure, with infiltration of both vocal cords and ulceration of posterior halves. Pronounced edema of both arytenoids. See Plate 7, Fig. 1.

Patient was very hoarse and complained of considerable dysphagia and dysphonia. Treatment: Artificial pneumothorax was attempted, but after the third or fourth insufflation patient became desperately sick with high fever and chills. This was apparently caused by partial spontaneous pneumothorax. Artificial compression was then discontinued, but was resumed several months later with extremely good result. Temperature became normal, cough decreased, while partial compression of the right lung is being maintained. See Plate 6, Fig. 2.

Larynx now shows only slight infiltration of posterior commissure, with healed ulceration of vocal process. Voice is normal and patient has no other symptoms referable to the larynx. See Plate 7, Fig. 2.

Case No. 5.—Th. LaR., age 29. Diagnosis: Pulmonary tuberculosis, far advanced. Tuberculosis of larynx, subacute type. Physical examination showed widespread infiltration of right lung with cavity in right upper lobe. Patient suffered from repeated hemoptysis. Examination of the larynx revealed

infiltration of posterior commissure with involvement of both vocal cords. See Plate 9, Fig. 1.

Patient was hoarse on and off during the previous year. Treatment: Artificial pneumothorax was induced with very good result. Hemoptysis was controlled after a partial collapse had been obtained. See Plate 8.

Laryngeal lesion cleared up entirely. See Plate 9, Fig. 2.

Patient was doing extremely well, having gained fifteen pounds in weight and became ambulant.

In February, 1925, patient had severe hemoptysis which was finally controlled by further compressing the right lung. However, he developed hypostatic pneumonia in uncollapsed lung and died February 23, 1925.

Case No. 6.—Case of far advanced laryngopulmonary tuberculosis treated by artificial pneumothorax for eight months with decided improvement in both lungs and larynx:

J. K., age 27, came under my care July 29, 1921. Physical examination revealed extensive involvement of right lung with cavity formation in right upper lobe and infiltration of left apex. See Plate 10, Fig. 1.

There was continuous fever in the afternoon and a great deal of cough and profuse expectoration. Larynx showed infiltration with papillomatous growth at posterior commissure with infiltration of both vocal processes. See Plate 11, Fig. 1. Patient was very hoarse.

Hygienic-dietetic treatment was given for two years without any effect. Artificial pneumothorax was, therefore, resorted to and patient showed remarkable improvement. After a few months of treatment a partial collapse of the lung was obtained. Cough and expectoration almost disappeared; temperature became normal.

Examination of larynx March 4, 1925, shows all structures to be normal, with exception of very slight thickening at posterior commissure. See Plate 11, Fig. 2. Voice is very good and slightly low pitched.

After eight months pneumothorax was discontinued, on account of adhesions.

Case No. 7.—Case of laryngopulmonary tuberculosis treated by artificial pneumothorax with no improvement. Patient died:

S. L., age 26. Diagnosis: Pulmonary tuberculosis, far ad-

vanced and active. Physical examination revealed widespread infiltration of right lung with cavity formation and softening in right upper lobe. Larynx showed infiltration and erosion of entire right vocal cord and infiltration with superficial erosion of posterior commissure. See Plate 12, Fig. 1. There was continuous afternoon fever with troublesome cough and blood streaked sputum.

After two months of observation artificial pneumothorax was resorted to and was continued for about seven months, with no amelioration of symptoms. Cavitation remained stationary. In December, 1923, it was decided to discontinue artificial compression. Patient became steadily worse and died a year later. Plate 2.

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2. Goodall, H. S.: Transactions, National Association for the Study and Prevention of Tuberculosis, 1914.

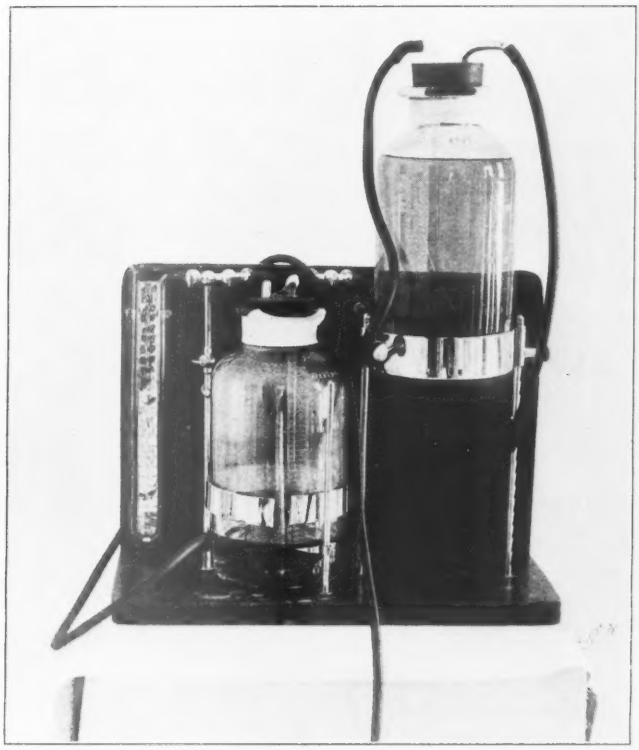

Fig. A.
Dr. Samuel Robinson's Artificial Pneumothorax Apparatus.

Fig. B.
Position of Patient.

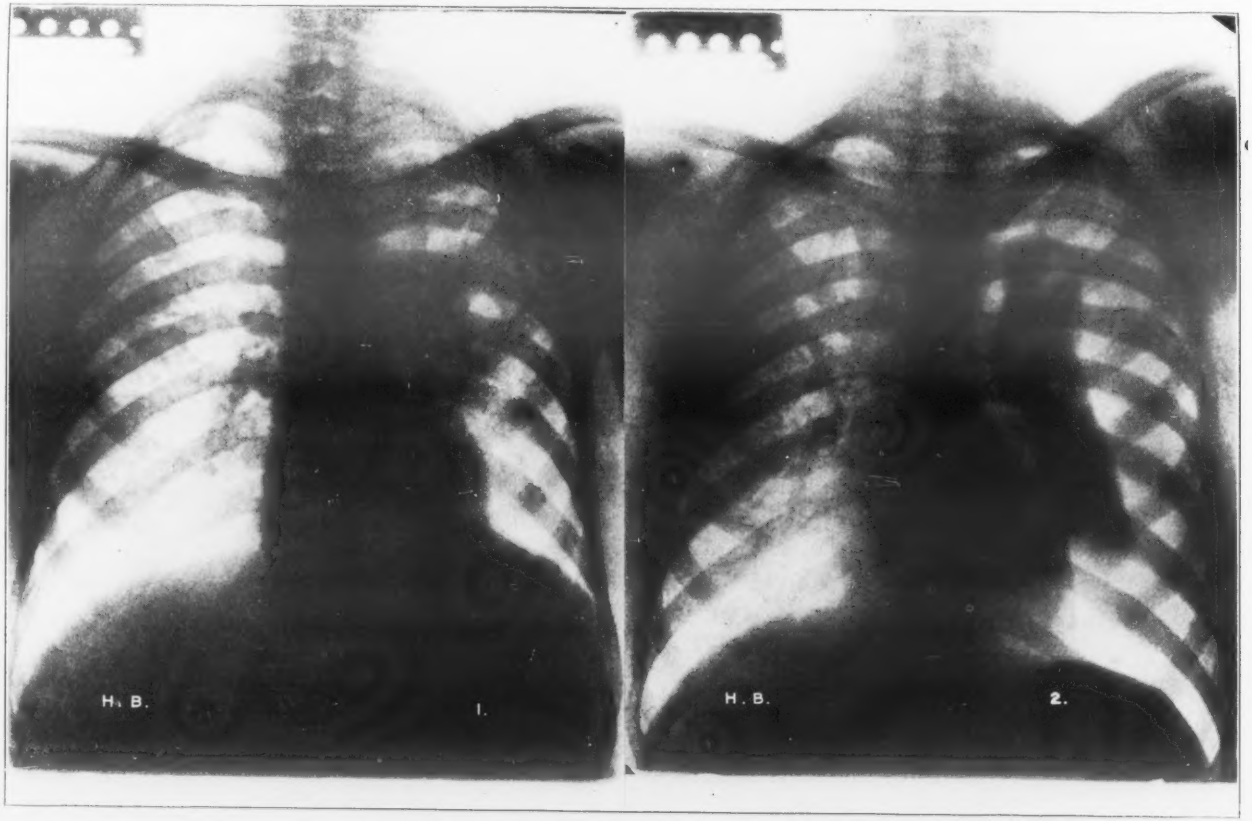

Plate 1. Case 1.

Far Advanced Pulmonary Tuberculosis, Active, confined to left lung.

Left lung 100% compressed by Artificial Pneumothorax.

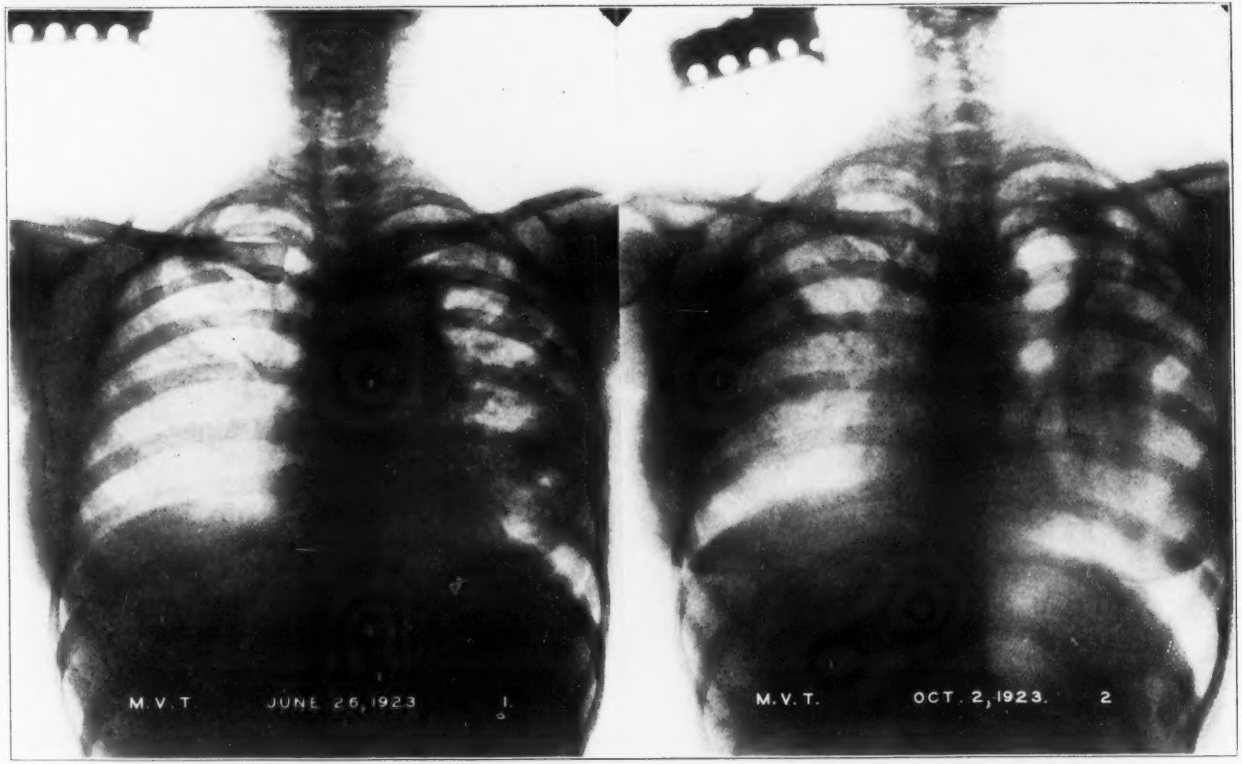

Plate 2. Case 2.

Far advanced tuberculosis with complete involvement left lung and cavity formation lower portion.

Partial compression left lung, complete compression being prevented by numerous adhesions.

and cavity formation lower portion.

vented by numerous adhesions.

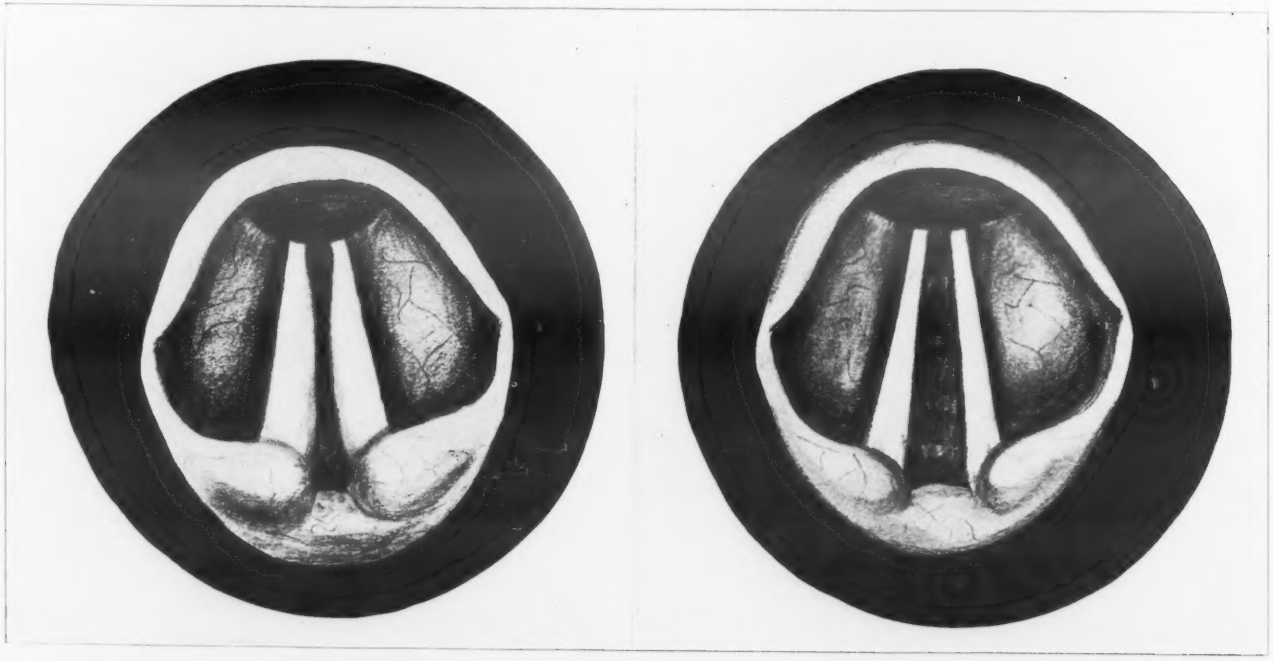

Plate 3. Case 2.

Acute infiltration of arytenoids and cords. Before treatment. After four months treatment by artificial pneumothorax. All structures almost normal.

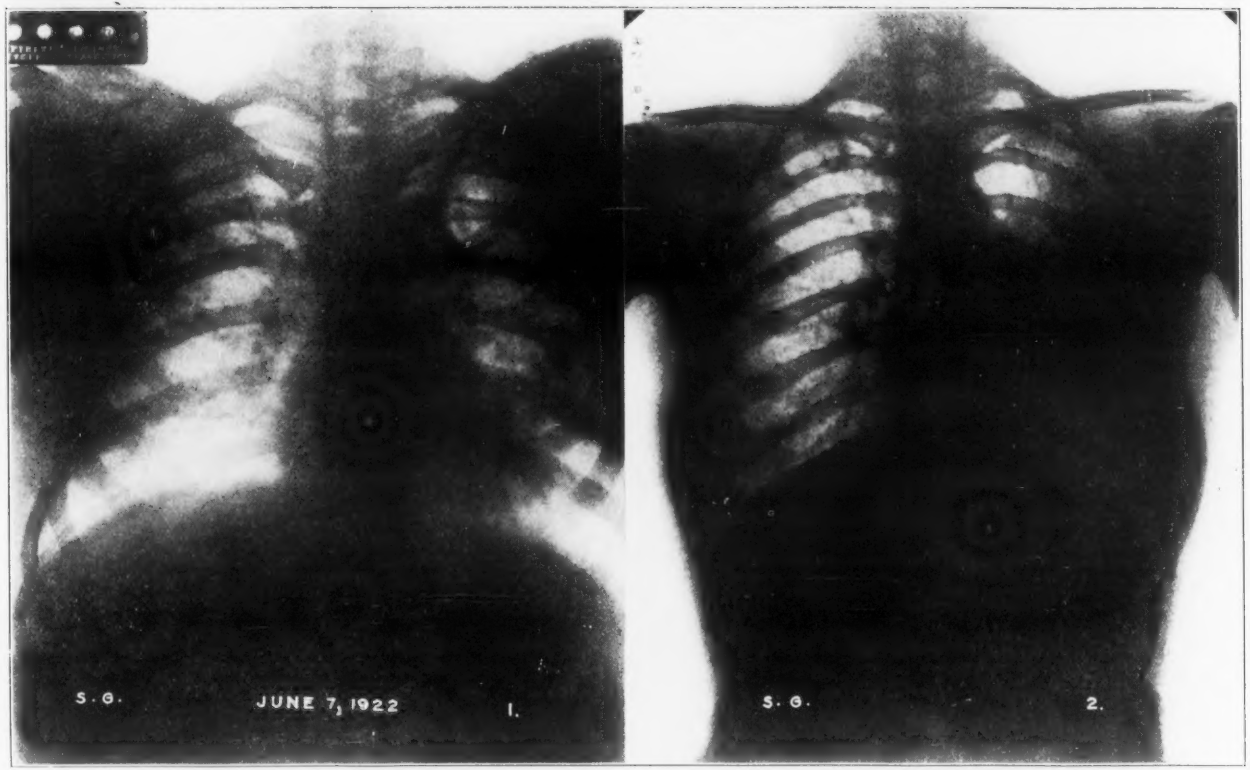

Plate 4. Case 3.

Widespread infiltration of left lung, with cavity formation left upper.

Organized hydrothorax, following artificial and spontaneous pneumothorax with pronounced amelioration.

Widespread infiltration of left lung, with cavity formation left upper.

Organized hydrothorax, following artificial and spontaneous pneumothorax with pronounced amelioration.

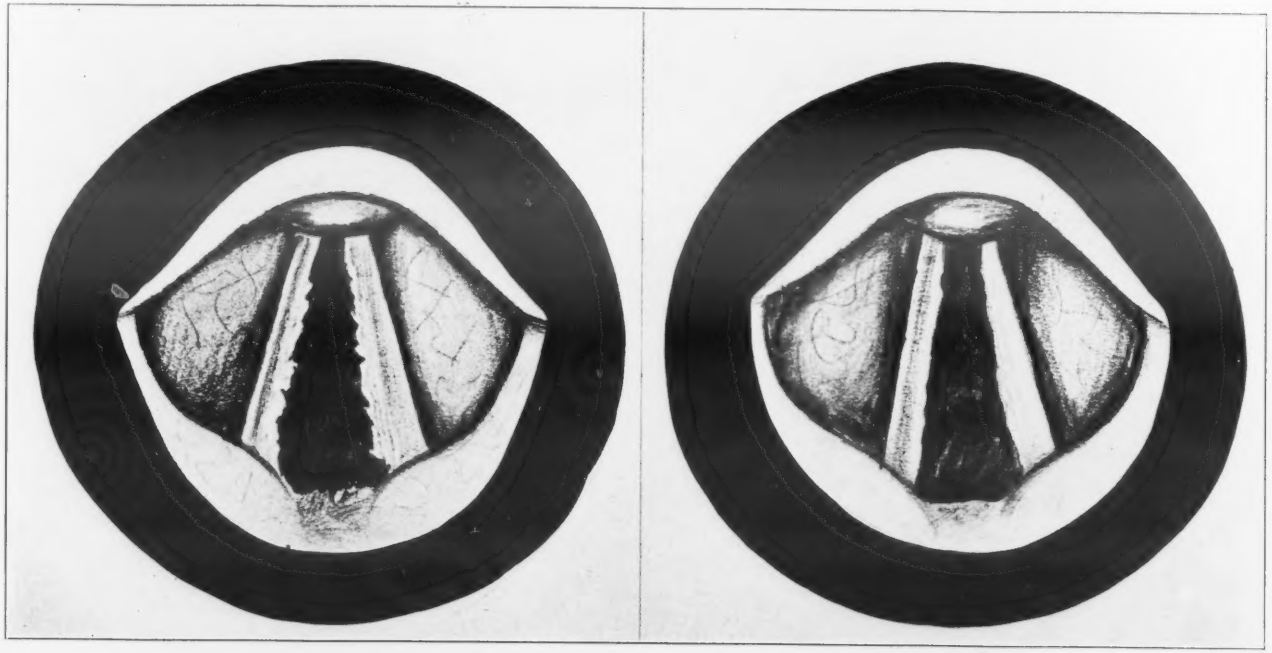

Plate 5. Case 3.

Before pneumothorax. Infiltration of both vocal cords with ulceration. Pronounced hoarseness.

Four months after pneumothorax. Laryngeal tissue improved. Hoarseness much lessened.

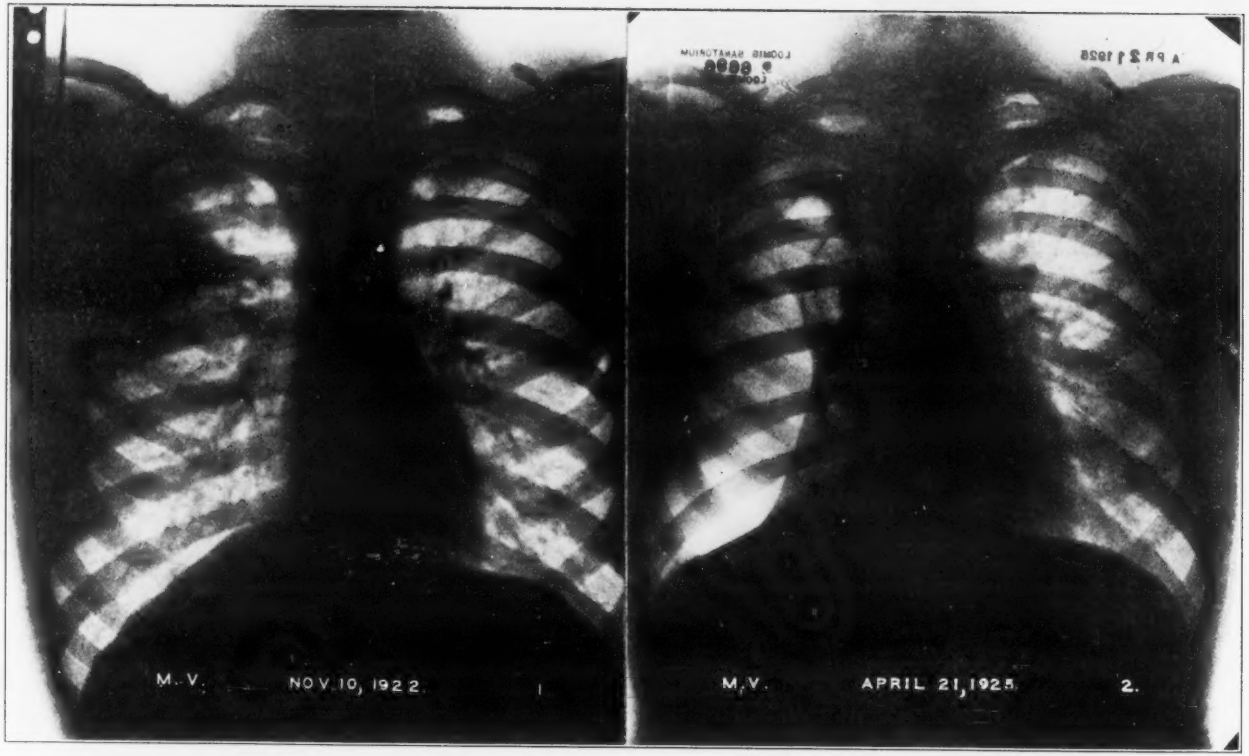

Plate 6. Case 4.

Complete involvement of right lung.

Partial pneumothorax, right lung, with compression, except upper lobe, which is held by adhesions.

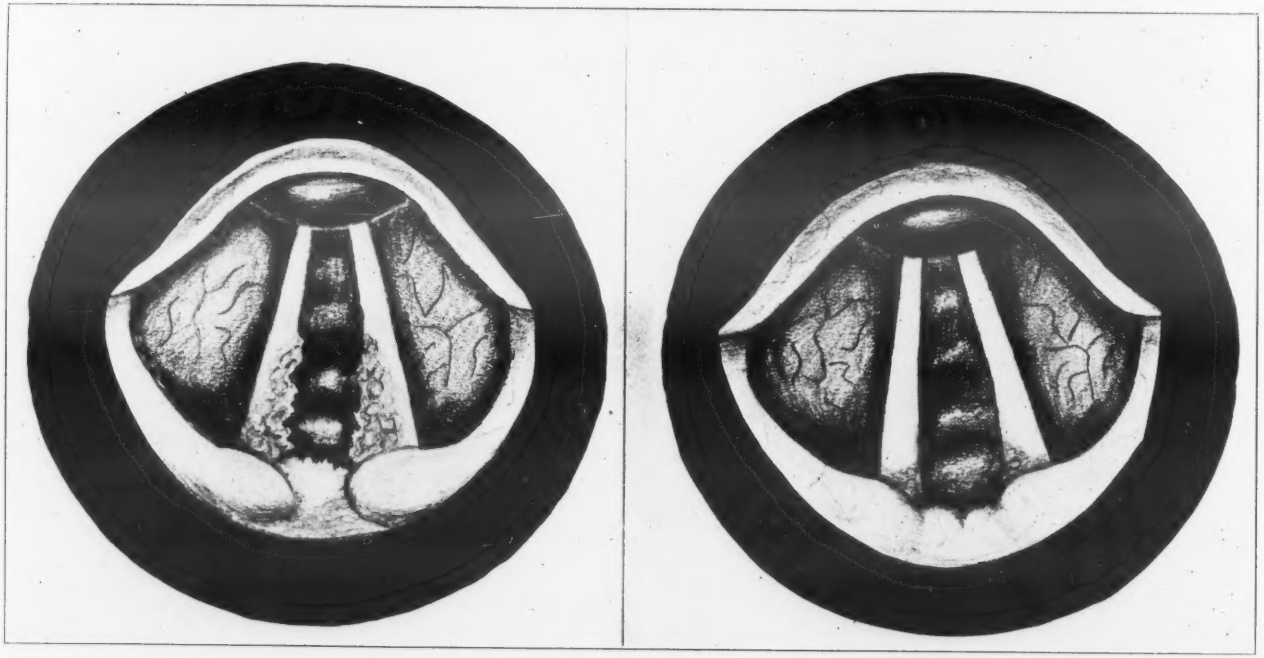

Plate 7. Case 4.

Larynx before treatment showing infiltration with ulceration of both cords and posterior commissure.

Several months after artificial pneumothorax begun. Larynx almost completely healed.

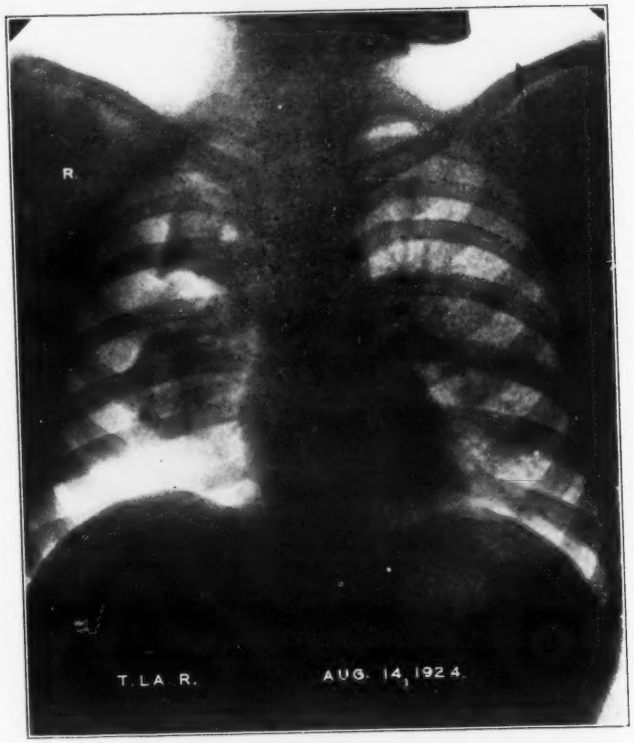

Plate 8. Case 5.
Advanced pulmonary tuberculosis, right lung compressed about 60%, with great symptomatic improvement.

60%, with great symptomatic improvement.

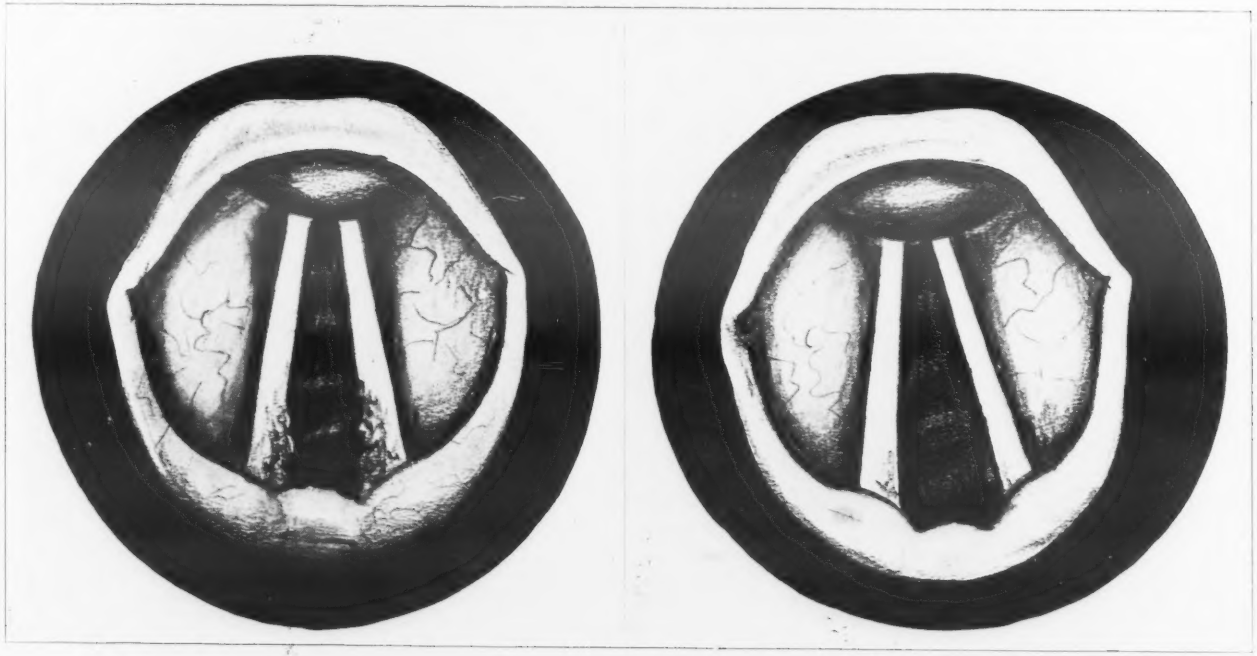

Plate 9. Case 5.

Larynx before treatment, showing ulceration of both cords and and posterior commissure.

After partial pneumothorax was obtained.

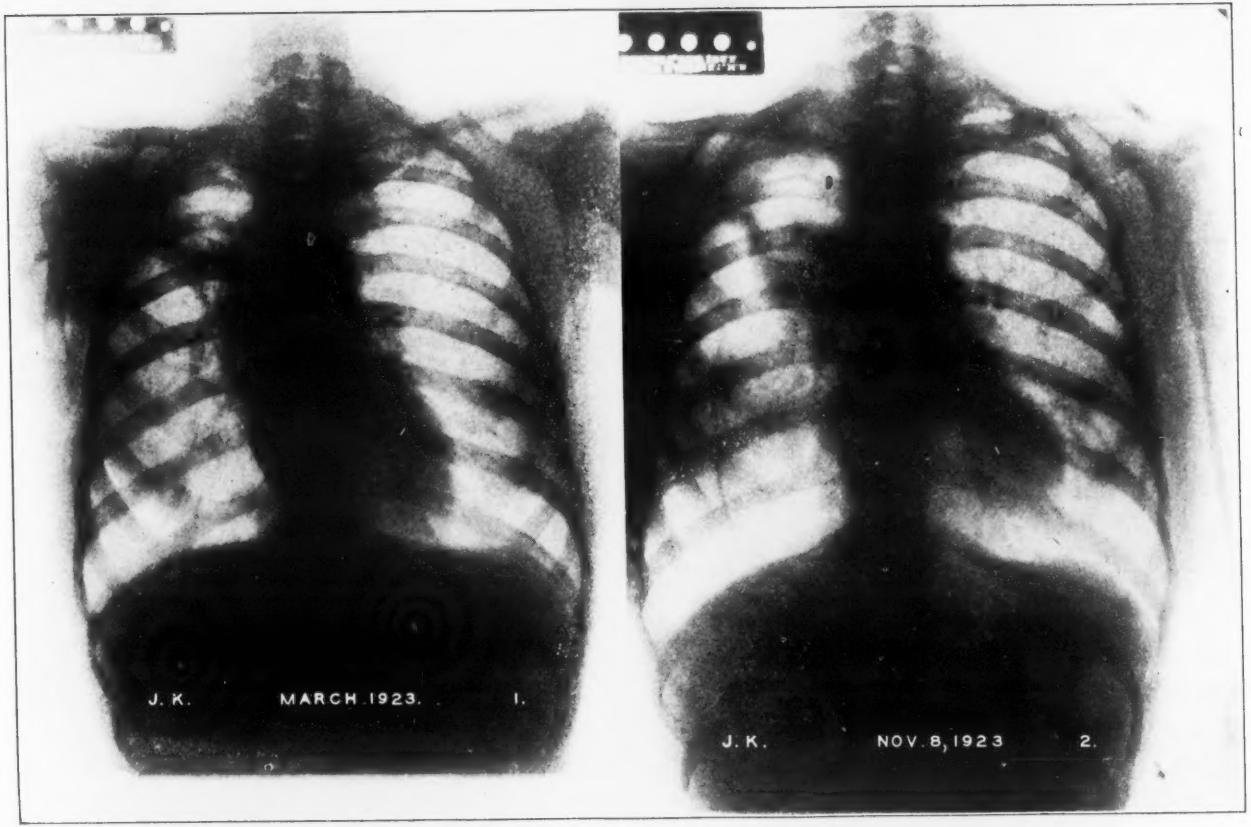

Plate 10. Case 6.

Widespread involvement or right lung. Partial pneumothorax with marked general improvement.

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Widespread involvement or right lung. Partial pneumothorax with marked general improvement.

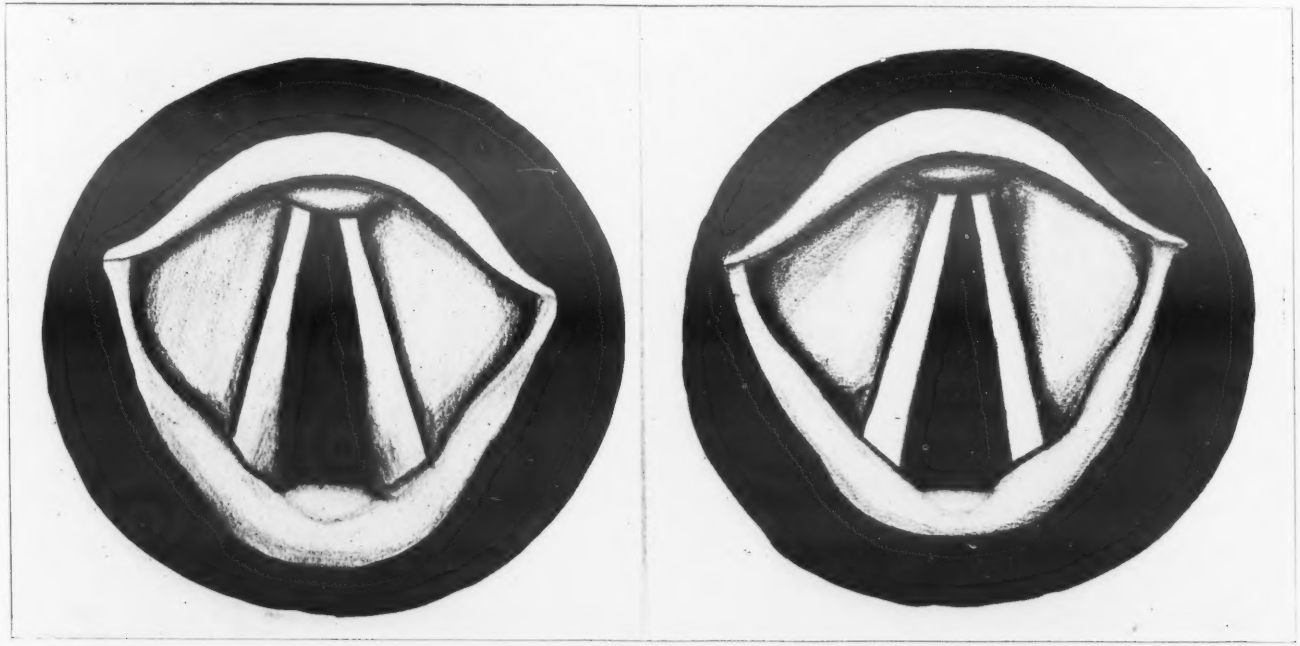


Plate 11. Case 6.

Before artificial pneumothorax treatment. After artificial pneumothorax treatment was well established.

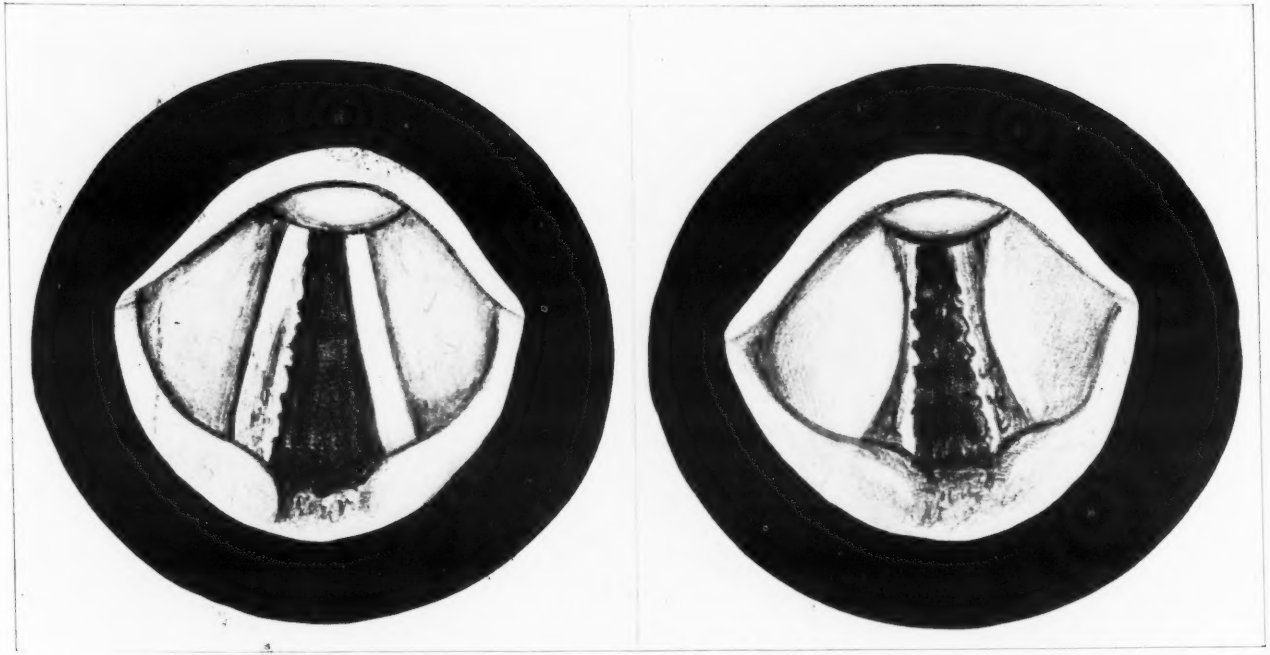


Plate 12. Case 7.

Advanced laryngopulmonary tuberculosis with widespread involvement of right lung, right vocal cord and posterior commissure.

Artificial pneumothorax failed to arrest pulmonary lesion; larynx remained the same and patient died a year later.

IV.

AN UNUSUAL ETHMOIDAL FINDING.

By S. R. Skillern, Jr., M. D.,

Philadelphia.

Mr. President and fellow members of the Philadelphia Laryngological Society and guests, I think I have a very unusual and unique case in medical history to present to you this evening, and if you will bear with me I shall first give you the case history and findings which have been of importance in reaching a conclusion.

History: P. F., age 56, Hebrew dry goods merchant, married, six living healthy children; wife living and well; no history of miscarriage. Past medical history: Hospitalized one week at the age of twenty years for stomach condition, otherwise always healthy. For the last two years has had an average of one head cold per month, otherwise well. Family history negative. Venereal history: This man denies absolutely any venereal condition.

Present illness began November 3, 1924, with cold in head and chest, for which he was attended by his family physician. His cough improved, but head condition became progressively worse, with the development of a heavy yellowish white discharge from the nose, chiefly on the right side. This discharge accumulated in his nose and throat during the night and, on account of its viscosity, he had great difficulty in ridding himself of it in the morning. He states he always felt much better after cleansing his nose and throat, but had a continual headache, which always became progressively worse during the day, and by night was very severe, seeming to center over his right eye and on the top of his head. He also complained of a cold sweat across the nape of his neck each evening.

Awaking the morning of November 12th, he found his right eye closed and was unable to lift the lid, but there was no pain, swelling or tenderness. Being greatly alarmed, he went at once to see his family physician, who sent him to the Wills Eye

Hospital, where he was hospitalized for examination and study. The findings obtained were "Ptosis of right eye, of syphilitic origin, a four plus Wassermann, probable sinusitis." The case was then referred to the Skillern Clinic of the Graduate School of the University of Pennsylvania on November 15th.

Examined in the outpatient department by Dr. George Whelan, the following was found: Both nasal fossæ were filled with yellowish white pus. After cleansing by blowing, the nasal cavities were so swollen and congested that an examination was impossible without shrinkage, so 10 per cent cocain was applied to the Schneiderian membrane. After shrinkage, both nasal cavities were irrigated with warm normal saline solution. Examination revealed pus coming down on both sides of the right middle turbinate and to a less extent between the left middle turbinate and lateral wall. The right middle turbinate had undergone polypoid degeneration. Septum markedly deflected to the left. A needle puncture of the right maxillary sinus showed spaghetti-like pus in the washings. The throat was red and congested, with infected tonsils and chronic pharyngitis, the posterior walls being streaked with yellowish white pus. The patient stated that his headache was much better following this examination. This patient was then told to report to the X-ray department the following day for examination.

On November 17th he was admitted to the hospital for operation and study and was assigned to me for demonstration to the graduate students in laryngology.

Examination: White male Hebrew, about 56 years of age, fairly well nourished, no apparent deformities. Head normal in size and shape. Eyes, complete ptosis of right eyelid; no edema, swelling or tenderness; no exophthalmia; left eye normal in all respects. Pupils normal in size and shape; slow reaction to light and accommodation. Muscular action of the right eye was normal to the left and downward, with a slow, jerky movement when rotating to the right, and no movement of the eye when attempting to look up. Diplopia in all of the left conjugate field; otherwise normal, but slight infraorbital pain elicited upon deep pressure. Nose normal in size and

shape. Mouth, teeth dirty and in poor condition. Throat, chronic infected tonsils. Pharyngitis with pus on the posterior wall. Laryngitis. Chest and abdomen and extremities reported normal, excepting for a possible slight increase in his reflexes.

Intranasal examination: Due to the hyperplasia and true hypertrophy of the inferior turbinates and generally swollen condition of the entire Schneiderian membrane, the nares were so occluded examination without shrinkage was impossible. After shrinkage, the right middle turbinate was found to be a degenerated polypoid mass with heavy yellowish white pus coming down over it. This pus traced upward to the ethmoid region and was seen extending backward into the choana. The right side showed the greater involvement. X-ray plates were diagnosed as purulent ethmoiditis, with a probable sphenoid involvement.

Procedure of operation: While the anesthesia was taking effect my brother gave the operating student minute blackboard instructions in the removal of the middle turbinate by the scissor-snare method. Then, under my personal supervision, the anterior two-thirds of the middle turbinate was removed. Examination of the turbinate showed a degenerated polypoid mass containing an ethmoid cell (socalled bone cyst) filled with pus. The operated field revealed pus coming down from all of the opened ethmoid cells. A small piece of the anterior end of the middle turbinate was seen in situ, and back, in the region of the posterior third, a fan shaped mass of degenerated mucous membrane could be observed. This degenerated mass was removed by the Luc forceps and the entire field swabbed dry with cotton applicators. I have carefully given all this more or less tiresome detail, which we all know, merely to show that no sharp or pointed instruments were used and that what is to follow could not have been caused by instrumentation at the time of the operation.

A small piece of the posterior end of the turbinate still remained, but as this was not interfering with drainage or obstructing the view of the anterior sphenoid wall it was not removed at this sitting.

While I was getting a sound my brother took the chair and attempted to sound the sphenoid sinus. After sponging the blood away from the posterior nares he found what he thought

to be the sphenoid ostium, 1 cm. higher than its normal position, being of a more or less irregular oval shape, about 3 mm. in size. A sound passed readily into this opening without meeting resistance of any sort. You may well imagine my surprise when, instead of striking the posterior wall of the sphenoid sinus at 8 or 9 cm., its usual depth, or even 11 cm. for a deep sinus, the probe continued to pass in, still without meeting any resistance until the entire length of the probe (7¾ inches) was buried in the skull. The only reason the probe did not pass farther into the skull was because the bent probe handle was resting against the lips, as shown by the photograph. During the passage of the probe and the entire time the probe was in the skull the patient did not complain of pain or manifest any nervous symptoms, as might be expected to occur with the stimulation of any of the motor or sensory centers in the brain or cord. The probe was removed and the nose examined without any apparent changes. In other words, there was no escape of cerebrospinal fluid or blood following the removal of the probe.

Dr. Ridpath next took the chair and passed the probe into the same cavity, with the same results. The probe was next passed by me and left in situ while the patient was placed in a wheel chair and taken to the X-ray rooms, while my brother, Dr. Ridpath and I discussed its probable location. I suggested a very deep sphenoid cavity, as I had once seen such a skull with a sphenoid cavity 12 cm. in depth from the nasal orifice. A lateral view under the fluoroscope showed the probe extending upward and backward from the nasal cavity, apparently through the brain substance, to within 2½ cm. of the inner table of the vertex of the skull. In a frontal view the probe was seen to be in the midline of the skull, probably in the median fissure.

While the necessary changes were being made for the taking of X-ray plates, I hurriedly went over the patient's major reflexes, which I found to be about normal. Questioning the patient, he stated that he felt all right, excepting the probe handle was pressing against his lip.

The pictures which I shall show you were then taken and the patient returned to the ward. A study of the X-ray pictures, showing the probe had passed into the skull above the

sphenoid sinus, through what was evidently a dehiscence in the posterior ethmoid capsule, or through an unusually large posterior ethmoid foramen, situated in the fovea ethmoidalis at its junction with the orbital plate of the frontal and lesser wing of the sphenoid. Normally the posterior ethmoid foramen is quite small and transmits the posterior ethmoid artery, which, as we know, supplied the posterior ethmoid cells. In a normal subject it would be impossible to pass a sound through this opening into the brain, due to its anatomic construction and size. The probe had passed backward and upward between the lobes of the brain, through the median fissure, until it had buried its full length. The only reason that I see why the inner table of the skull was not reached was because the probe was too short to reach it.

When the patient was seen one hour later, he complained of a mild headache. Temperature normal. The following day no change was noted in the patient's condition, heavy purulent discharge from the nose persisting.

Two days later, on the 19th, the temperature had risen to 102, pulse 90, and respiration 32 at 2 p. m. The blood count, taken that morning, was: Erythrocytes 4,860,000, leucocytes 13,000, hemoglobin 75 per cent. Differential count: Small lymphocytes 10, transitional 1, polynuclear 88, eosinophiles 1. Blood culture, no growth. Wassermann reports, by Doctors Kilmer and Yagle, negative. Urine negative. You will recall that the Wills Eye Wassermann was 4 plus. The patient was taken to the operating room for examination and treatment. Eye muscles showed marked improvement; no Babinski, ankle clonus or Kernig's sign present. Knee jerk about normal.

Intranasal examination showed heavy purulent discharge, which was removed by sterile hot normal saline solution; inferior turbinates, shrunken with cocain; small polypoid mass seen in posterior ethmoid region removed with Luc forceps. Both nasal fossæ were well irrigated with warm normal saline solution until the washings returned clear. The first washings showed quantities of pus and shreddy degenerated epithelium and polypoid tissue. After this irrigation the patient stated he felt better than he had felt since he was taken sick, and, as will be shown, his condition steadily improved until he was discharged from the hospital, December 29, 1924.

Following the above irrigation, the patient's temperature dropped from 102.4 to 101.4 degrees, and, with the exception of a rise of .3 degree at 6 p. m., it continued to drop to 97.3° at 11 p. m. At 3 a. m., on the 20th, it reached normal, where it remained until his discharge.

His blood count on the 20th showed a loss of 400,000 erythrocytes, 1,000 leucocytes and 10 per cent hemoglobin. The only internal medication given, until December 24th, was calomel, magnesium sulphate, urotropin, aspirin and digitalis. I mention this fact, as neosalvarsan was started the afternoon of the 24th.

Awaking the morning of the 24th, the patient found he could open his right eye about a quarter of an inch. This steadily improved until the 28th, when the patient had full control of not only the levator palpebræ, but all the eye muscles as well. All diplopia had disappeared, and his pupils reacted normally to light and accommodation, and, as he stated, all headaches and pain had left him, he was discharged on December 29th with instructions to return for further treatment to the outpatient department. This treatment consisted of irrigations with normal saline solution, and the ethmoid region packed with cotton saturated with 20 per cent neosilvol solution for one hour, the treatment being carried on triweekly until January 5th, with little or no improvement of the purulent ethmoid and sphenoiditis, so that on January 7th I had the patient report to me in clinic. I removed the remaining portion of the posterior end of the middle turbinate, which had become polypoid, and opened the bulla and the anterior ethmoid cells in back of it. With fear and trembling in my heart, I then probed the sphenoid ostium and found the sphenoid filled with polypi and pus, so with a small sharp hook I enlarged the ostium downward. This seemed to be of marked benefit to the patient. As his purulent condition began improving, the pus gradually diminished under irrigations with normal saline solution.

The patient reported to me January 11th, stating he was feeling fine, no more headaches or trouble with his eyes, good appetite, was gaining in weight and had a good, healthy color to his skin. Dr. Whelan reported his nasal condition much

improved, but still a good deal of pus coming away. The next time I saw this patient was the afternoon of January 19th, but as this is another story I shall speak of it later.

My reasons for reporting this case are twofold: First, and mainly, I think it a very unusual one, as I know of no other case in which a probe has been passed through an infected nose up into and apparently between the lobes of a man's brain, until the inner table of the skull at its vertex was nearly reached, without signs or symptoms of any kind at the time or meningitis developing afterwards. In fact, I can find nothing in the medical or surgical histories of a probe ever having been passed through the nose into the living brain. Secondly, that the average physician, and I include a good many ophthalmologists, are apt to say "syphilis" as soon as they see a patient with ptosis and no other acute general symptoms. I say this because I gave this man's symptoms to several physicians, among whom were three ophthalmologists, and as soon as I said ptosis they looked wise and said, "sure syphilis." I do not believe this man's eye condition was due to syphilis. I do not say he did not have syphilis, as we have a four plus Wassermann report from one institution and a negative one a few days later from another laboratory, but I will say I could not find any symptoms of syphilis. If this man's ptosis was of syphilitic origin, would the operation have been a benefit to him, and would his paralyzed muscles have regained their normal function? Most certainly not! In my opinion, this man's eye condition was due entirely to the toxins from the ethmoid and sphenoid. Although the purulent ethmoiditis had not reached the stage of actual rupture into the eye or brain, it had so involved the surrounding bone and tissues that there was a leakage of the toxins, either into the orbital space through the lamina papyracea, which from its name alone we know to be a very thin plate of bone, or into the brain by extending backward in the bony channels of the blood vessels or lymphatics, or, as Dr. Samuel Kulkin of New York, in his article "Case of Intranasal Ethmoid Exenteration Accompanied by Uncontrollable Hemorrhage, Death," in the January issue of the Laryngoscope, quoting Dunbar Roy, from the ANNALS OF OTOLOGY, RHINOLOGY AND LARYNGOLOGY states, "Evidently a slow bacteremia had been progressing for some

time, due to the absorption for years of pus from the ethmoid cells, and this had undermined the coats of the blood vessels. These extreme cases of necrosing ethmoiditis are not without their dangers," or, quoting from Skillern's book, page 13, he states, "The olfactory nerves, during their passage through the cribriform plate, are enveloped in a prolongation of dura mater, which connects more or less intimately with the nasal mucosa. This anatomic formation favors the ready transmission of infection from the nasal cavity into the meninges." I speak of this infection of the coats of the blood vessels and into the brain, not that I believe his eye condition was of a central origin, but for reasons I shall bring out later.

My belief is that the toxins, by extending through the lamina papyracea, had either paralyzed the muscles of the eye or had cut their motor impulses from the brain by a paralysis of the nerve terminals in or in close proximity to the muscles. My understanding of the third nerve palsy is that all the muscles governed by this nerve are paralyzed. If this be true, we did not have a nerve palsy, as only the levator palpebræ and inferior oblique were involved, the remainder acting normally. Then, too, we must take into consideration that the external rectus muscle was also involved, as shown by the slow, jerky movements of the eyeball in attempting to rotate outward. The muscle, as we know, is independent of the third nerve, being governed by the sixth.

Let us consider for a moment the origin of these muscles. The levator palpebræ superioris arises from the lesser wing of the sphenoid, above and in front of the optic foramen. The inferior oblique arises from a depression in the orbital plate of the superior maxillary, external to the lacrimal groove and the external rectus, from two heads, the first, from the outer margin of the optic foramen, the other from the ligament of Zinn. It is possible but hardly probable that the leakage of toxins through the lamina papyracea would seek out these muscles alone, when the other eye muscles, which have the same origin or an even closer origin to the seat of the disease, remained normal. Consequently, my deductions favor a nerve toxemia, rather than a muscle involvement, especially so when we consider the close proximity of the nerves.

Now let us go back a moment to our operation. As soon as proper drainage was effected, this man's condition began to improve. Why? Because he no longer had toxins under pressure and, as all fluids do, seek the line of least resistance, the toxins drained off as they formed, with a consequent daily improvement of not only his eye condition but his general health.

Now comes the sad part of the story, and what to me was a great disappointment, as I had hoped to have this patient here tonight, that you gentlemen might see him with your own eyes.

The afternoon of the 19th, I was told of his being in the hospital. I found him tossing about in the bed in agony from what he described as a bursting headache, which one-half grain of morphia did not relieve. His pulse was slow and bounding, and out of ratio to his rapid respirations. His eyes were normal in appearance and reflexes greatly exaggerated. He stated he had been feeling better than he had for years until the evening of the 18th, when he ate a large meal of meat. This, he stated, gave him a headache, which had been getting worse by the minute, and he begged for relief. A spinal puncture showed very bloody fluid, not under pressure. At 11 p. m. he was in a comatose condition and he died at 6 a. m., January 20th.

A necropsy by Dr. Eugene Case, whose report is as follows: "The body is that of a well developed man of middle age. The pupils are unequal, the right being contracted. The bones of the skull are not thickened and no difficulty was experienced in removing the calvarium. No fractures present. After the removal of the brain, a search was made for an abnormal opening in the ethmoid or sphenoid bone and the overlying dura on the right side, for during life a probe introduced into the right nostril had passed upward into the cavity of the skull between the cerebral hemispheres to a point about 2 cm. from the vertex. No abnormal opening was found, however, and an attempt to repeat this procedure on the autopsy table failed, though a probe introduced into the right posterior ethmoid foramen passed into the nasal cavity and emerged from the right nostril. The same procedure was successfully

but less readily repeated on the left side. An inspection of the ethmoid and sphenoid sinuses of the right sides revealed a thickened, edematous mucosa, bathed in pus. The right frontal sinuses also contained pus, but those on the left side appeared to be clear. Cultures yielded growths of nonhemolytic streptococcus, the pneumococcus, staphylococcus albus, a diphtheroid and a bacillus of the Friedlander group. No evidence of a recent or old meningitis was found. The brain was bathed in considerable blood, which escaped when this organ was removed. After hardening, the brain weighed 1,770 grams. The surface is very hemorrhagic, and in the right cerebral hemisphere there is a softened area with a depressed surface of irregular outline and occupying a portion of the parietal and occipital lobes, especially the posterior cerebral gyrus, the superior parietal lobule and the inferior parietal lobule. The pia arachnoid over this area is thickened, the vessels are prominent and the convolutions smaller than the corresponding ones on the opposite side. A cut section of this area reveals a group of telangiectatic blood vessels with relatively thin walls and filled with clotted blood. This grouping is more or less circumscribed, suggesting an hemangioma of the cavernous type. Other sections of the brain show hemorrhage into the substance of this hemisphere, filling the entire ventricular system, and the escape of blood into the subarachnoid and subdural spaces. The vessels of the brain show little or no sclerosis.

"Microscopy.—Sections from the area of telangiectasis show blood spaces of good size with fibrous walls, forming a vascular network similar to cavernous hemangiomas elsewhere. The mucosa of the sinuses was the seat of a chronic inflammation. Diagnosis: Chronic purulent sinusitis of the ethmoid and sphenoid sinuses of the right side of the right frontal sinuses. Cavernous hemangioma of the right cerebral hemisphere. Hemorrhage into the right cerebral hemisphere, the entire ventricular system, and the subarachnoid and subdural spaces."

While the body was on the table I attempted (after cleansing the nose with dry cotton and seeing pus welling out of the sphenoid) to sound the opening through which the probe had passed. After many repeated attempts with failure I had Dr.

Whelan try, but without success, so the skull was opened, about twenty ounces of blood escaping, thus confirming in my mind a tentative diagnosis of cerebral hemorrhage. The brain showed no signs of injury or meningitis, nor did the meninges, through which the probe had apparently passed. In fact, they both appeared to be normal to macroscopic examination.

Now comes an interesting detail. A minute examination of the dura covering the entire fovea ethmoidalis failed to show any sign of perforation or injury. When this was stripped off, the underlying bone was of a darker bluish color than normal, but search as I would, I could not find where the probe had passed through any opening, from the cribriform plate to the sella turcica, excepting the posterior ethmoidal foramen. Removing the fovea ethmoidalis and opening the sphenoid showed degenerated polypoid purulent masses on the right side. The right frontal and left ethmoidal cells showed a hyperplastic condition of the mucous membrane, the left frontal and sphenoid being normal. The question now arises, what happened to the dehiscence of bone through which the probe passed. From the color and general appearance of the bony plate covering the ethmoid cells, it was diseased throughout, and yet apparently Nature had closed over the original bony dehiscence, if one was present at the time of the operation.

I know this paper has been long, but I hope not very tiresome. The reading of the routine history, operations, etc., is necessary, in my opinion, to bring out this point.

The moral to be deduced is, do not diagnose all ptosis cases, even though they may appear to be of frank syphilitic origin, as syphilis, until a thorough intranasal examination has been made by a competent sinuologist and all sinusitis ruled out.

It is my hope that this case may be discussed fully that I may acquire a greater knowledge of such cases and learn of any similar cases in rhinologic history.

Fig 1

Fig. 2.

Fig. 3.

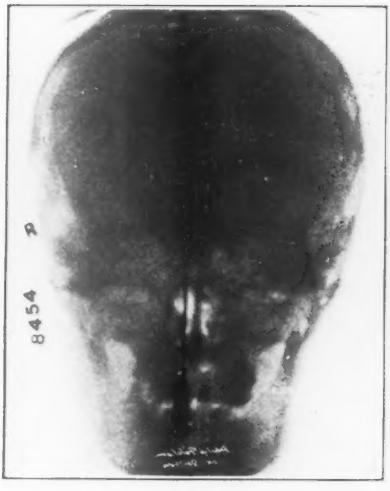


Fig. 4.

Fig. 5.

V.

THE MUCOUS GLANDS OF THE PALATE IN RELATION TO THE UPPER POLE OF THE TONSIL.*

By Harry L. Baum, M. D.,

Denver.

These observations are the result of investigations made by the author in an effort to explain certain clinical findings met in the daily routine of tonsil surgery which, though minor, may be admitted to be worthy of your consideration in view of the fact that it is the summation of an infinite number of details which makes for perfection in the quality of our work.

You have doubtless observed that following the removal of tonsils, by whatever method, there is frequently an apparent roughening to be seen in the superior portion of the tonsillar fossa while the remainder of the fossa is smooth and the aponeurosis of the muscles intact. At this point in the fossa there may at times be some irregular masses of glandular tissue extending into the space just vacated by the upper pole of the tonsil. This appearance may frequently give rise to doubt in the mind of the operator as to whether all lymphoid tissue has been removed from the region, and I have known surgeons, in their zeal, to follow this glandular tissue into the palate to such depth as to result in marked contracture. In such cases they were not removing lymphoid tissue, as you all know, but were invading the palate proper and removing its mucous glands. It is this relation of the mucous glands of the palate to the upper pole of the tonsil that I have set out to demonstrate, and to that end will call your attention briefly to some anatomic facts relative to it.

The glandular layer of the palate is in places as much as five millimeters in thickness and is situated between the fibromuscular layer above and the oral mucous membrane below. Laterally and below, in the region of the upper pole of the tonsil,

*Read before the Mid-Western Section of the American Laryngological, Rhinological and Otological Society, Colorado Springs, Colo., February 14, 1925.

the glandular masses thin out somewhat and become fewer in number, with more areolar supporting tissue. In this location the palatoglossus muscle is interposed between them and the mucosa, but elsewhere they are separated from the oral cavity by the mucous membrane only.

The pharyngeal aponeurosis, the real framework of the pharynx, is a distinct fibrous membrane lying between the muscular layer and the mucosa, and separating the tonsil from the superior constrictor muscle. Although the tonsil is nominally spoken of as resting on the superior constrictor, its bed is really formed by the aponeurosis, to which it is attached by a comparatively few fibers of loose areolar tissue and by the blood vessels passing to and from the tonsil and thus forming an additional bond of union.

The tonsil is bounded anteriorly by the palatoglossus muscle, arising from near the middle line of the oral side of the lower part of the soft palate and curving downward in the anterior pillar to the tongue.

Posteriorly, the palatopharyngeus muscle bounds it. Arising mostly from the palatal aponeurosis and the edge of the hard palate above the glandular layer, it curves downward in the posterior pillar to insert in the pharynx. From both of these the tonsil is separated by their aponeuroses, to which it is attached in a manner similar to its attachment to that of the superior constrictor.

Below, the lower pole of the tonsil blends with the lymphoid tissue of the base of the tongue.

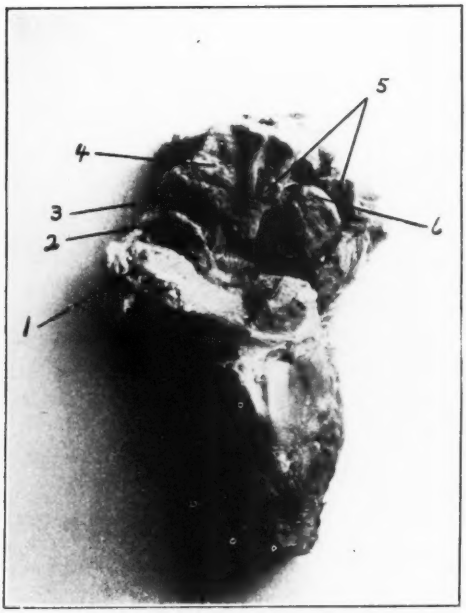
Above, its upper pole extends into a space in the palate containing only loose areolar tissue and mucous glands, from which it is separated by its own fibrous investiture only.

The tonsillar fossa is lined with a definite aponeurotic membrane anteriorly, posteriorly, externally and below, because the aponeurotic coverings of the muscles forming the fossa act as a lining for it. Above, however, the upper pole does not rest in a muscular bed, but extends slightly toward the midline and into the areolar tissue of the palate. The pharyngeal aponeurosis follows the superior constrictor upward, sending only a few very delicate fibers over the upper pole of the tonsil, thus leaving this portion of the fossa without an aponeurotic lining

after the removal of the tonsil with its own investing membrane. For this reason the superior portion presents an appearance different from that of the rest of the fossa, and may at times contain small projections of glandular tissue from the mucous glands of the palate. These masses may suggest the possibility of the presence in this location of a remnant of lymphoid tissue, but an attempt to remove them will quickly show the experienced operator that he is not dealing with lymphoid tissue, but with mucous glands, the pursuit of which would result in the virtual splitting of the palate.

To summarize, we have in the palate and lying between the palatoglossus and the palatopharyngeus muscles and internal to the superior constrictor of the pharynx, a space containing areolar tissue and mucous glands in close relation to the upper pole of the tonsil. This region is of importance to us in two ways: First, tonsillectomy opens this space, necessarily, and permits access to the glandular layer of the palate. Second, peritonsillar infection, occurring as it does between the fibrous covering of the tonsil and aponeurotic lining of the fossa, will, in many instances, following the line of least resistance, extend upward into this space above and around the upper pole of the tonsil. Here, forming pus, it tends to point through the thin mucous membrane of the palate above the origin of the palatoglossus muscle.

264 METROPOLITAN BLDG.



Dissection showing relation of upper pole of tonsil to palatal structures.

1. Mucous membrane of right side of palate and tonsil dissected away and turned downward.
2. Upper pole of tonsil lifted out of its bed in palate.
3. Cut edge of palatoglossus muscle.
4. Glandular layer of palate.
5. Cut ends of palatoglossus muscle turned back to show relation of upper pole of tonsil to palatal structures.
6. Upper pole of tonsil in normal position.

VI.

AURAL QUACKERY AND PSEUDOMEDICINE.

By Arthur J. Cramp, M. D.,

Director, Bureau of Investigation, American Medical Association,

Chicago.

There are styles in quackery. The prevailing modes of the current season are those products sold for the alleged sexual rejuvenation of the male. The products themselves differ widely in composition, ranging from testicular substance of ringtailed monkeys to radium emanations dissolved in drinking water. But in quackery, as in other well established branches of human activity, there are certain old standbys that, like Tennyson's Brook, go on forever. Of such are the consumption cures and the cancer cures; the epilepsy cures and the rupture cures; the female weakness cures and the weak men cures; the cures for fatness and the cures for leanness; the quacks who will fit glasses on no other data than your age and ability to pay the bill, and the faker who will sweepingly promise to cure deafness of whatever origin.

Classification.—What I have to offer this evening is a brief excursion into the realm of aural quackery and pseudomedicine. Considering the strictly specialized nature of the field, the proportion of deafness cure quacks to the total number of medical fakers is large. Many of them do business through the United States mails, although there are some itinerants who specialize in this branch. A few dispose of their worthless preparations through the retail drug stores. The mail order quacks may be divided into two classes: those who sell a more or less elaborate but worthless "course of treatment" and those, probably in the majority, who dispose of trivial, worthless and often dangerous devices at exorbitant prices. For the purposes of this talk the field has been arbitrarily divided into three groups: medicinal preparations, mechanical devices, and practitioners.

MEDICINAL PREPARATIONS.

One of the most widely advertised deafness cures of the medicinal type is that exploited by A. O. Leonard of New York City. A collection of Leonard's series of follow-up letters show them to be of the usual medical mail order type with stock testimonials, "bulletins" and other paraphernalia of the long distance quack. It seems that Leonard, like practically all mail order charlatans, sells the original letters he receives from victims and prospective victims. At least such letters by the hundred have been offered for sale and rent.

Leonard Ear Oil.—Leonard's Ear Oil has been sold not only on the mail order plan but also through the retail drug trade, for, unfortunately, some druggists are not above participating in the profits of medical fakery. A few years ago the Department of Health of the city of New York

Look for this Sign in
Drug Store
Windows.
It is a
LEONARD
EAR OIL
AGENCY
If your druggist is not an agent write me, 150 5th Ave., Suite 816, New York City, for descriptive circular and list of New York, Brooklyn and N. J. druggists who are agencies for Leonard Ear Oil. NO OIL SOLD BY ME AT RETAIL.

analyzed Leonard's Ear Oil and reported that it was, in effect, an emulsion of mineral oil and soft soap with a little camphor and eucalyptol. Leonard was arrested and found guilty of making false and misleading claims. He was sentenced to pay a fine of $250 or go to jail. At the same time the New York Health Department notified the druggists of that city that the continued sale of Leonard Ear Oil would make them liable to prosecution.

Later, Cleveland, Ohio, through its Commissioner of Health, declared Leonard's Ear Oil a misbranded product, and notified the druggists of Cleveland to discontinue the sale of the preparation. Still later the federal authorities seized a quantity of the Leonard Ear Oil, charging that the claim that the stuff was an effective remedy for the cure of deafness was false and

fraudulent, since the preparation contained no ingredient or combination of ingredients capable of producing the effects claimed. The court pronounced judgment of condemnation and forfeiture, and ordered the product to be destroyed. At the present time Leonard is advertising some ear drums and in connection with them exploits his oil.

Aurine Ear Balsam.—Another "deafness cure" of the medicinal type is a local product, Aurine Ear Balsam, put out by the Aurine Remedy Company. A few drops of Aurine Ear Balsam, it is claimed, will cause deafness of catarrhal origin to disappear. When there is a discharge from the ears the daily use of Aurine Ear Balsam will cause the discharge to stop. The deafness of old age can also be remedied by Aurine Balsam. According to the Aurine school of otology, head noises are caused by mucus that holds the small bones in the ear in one place; Aurine Ear Balsam loosens the mucus and the head noises stop. Thickened, relaxed or shrunken drums are due to the eustachian tubes becoming stuffed with mucus. A few drops of the "balsam" will soon restore the drums to their natural condition.

The chief stock in trade of the Aurine concern is testimonials, whose trend is to impress on the sufferer the idea that Aurine Ear Balsam will cure deafness and other ear troubles. Prospective victims are urged to send in $2 for a package. Those who bite get a 1 ounce bottle of oil—the "balsam"—an elongated toothpick with a pledget of cotton on one end, a medicine dropper, and a small wad of absorbent cotton. They are told to instill three or four drops of the Aurine Ear Balsam into each affected ear once a day and to clean the external meatus every other morning with the wooden swab. Their conception of surgical cleanliness is revealed in the suggestion that "it is a good idea to put fresh cotton on the ear swab each time it is used." In cases of discharging ears the victim is told to mix five drops of Aurine Ear Balsam with four tablespoonfuls of warm water and inject it into the ear with the glass dropper every other day. The victim is urged, "Stick to Aurine Ear Balsam and get rid of your trouble."

A preliminary examination of Aurine Ear Balsam was made in the chemical laboratory of the American Medical Association, and the chemists reported that the product appears to con-

sist largely of glycerin with boric acid, aromatic oils and what apparently is a local anesthetic.

Newspaper advertisements urge sufferers from deafness, head noises, discharging ears and all ear drum troubles to write to the Aurine Remedy Company for free advice. Those who do write receive a stock letter and a booklet entitled "How to Take Care of the Ears." The booklet describes the alleged origin of Aurine Ear Balsam. From this we learn that it was first made 25 years ago by Charles G. Foucek, president of the Aurine Remedy Company. We are told, further, that Foucek is a member of the Board of Pharmacy of the Department of Registration and Education for the State of Illinois. It seems that for over thirty years Foucek owned several drug stores, which he recently sold so that he could devote all of his time to this business.

The booklet also gives the experiences of Milton W. Folds, the manager of the company, who is said to have had ear trouble for fifteen years that resulted in deafness. He tried everything, lost faith in doctors and medicine, then used Aurine Ear Balsam and his troubles disappeared! According to Folds, nearly 100,000 men and women have used Aurine Ear Balsam with perfect satisfaction for every known ear trouble. In common with other mail order quacks, the Aurine Remedy Company publishes what it calls a legally binding guarantee to refund upon demand the purchase price, without question or delay, to anyone who is not entirely satisfied with Aurine Ear Balsam. We have in our files a letter from a poor dupe who wrote to the Mayor of Chicago telling him that, being hard of hearing, he had purchased Aurine Ear Balsam, and used it, with the result that the stuff made him stone deaf. He said that, although he had written to the Aurine concern, asking them to refund the money, he got no satisfaction.

Hearwell Oil.—Another deafness cure nostrum is "Hearwell Oil," put out by a Milwaukee concern. With the oil there also come some tablets to be used as a supplementary treatment. When subjected to preliminary tests in the association's laboratory the chemists reported that apparently Hearwell Ear Oil is crude Japanese camphor oil, a by-product in the manufacture of camphor, and the tablets sodium iodid in an excipient of elm bark.

Happy Letters Tell Wonders Aurine Ear Balsam is Doing

Here are a few letters telling of the wonderful results people have obtained from using Aurine Ear Balsam, — They are happy letters from happy people. Send for your bottle of Aurine today and I am sure you too will write a letter telling how happy you are that Aurine Ear Balsam helped you.

HEAD PAINS HAVE CEASED.

Hoboken, N. J., Aug. 6, 1924.

Dear Sir: — Please send me another bottle of your Aurine Ear Balsam at an early date, as I have only a small amount on hand. I have been greatly benefited with what I have already used, and I feel with its continued use it will do all you claim for it. The head pains that I have suffered for so long ceased altogether and my hearing has improved to some extent, which is more than I had hoped for as my case is one of many years standing. I hope to have more good news for you in the next letter. You may send it C. O. D. as you did the last bottle.

Respectfully,
Mr. JOHN ROBERTS.

MARVELOUS RESULTS.

Los Angeles, Calif., Sept. 15, 1924

Dear Sir: — I have two friends who suffer with ear trouble and I am anxious for you to send them some of your literature. I received marvelous results from your Ear Balsam and want these friends to try it. I will also write them personally about it. I have only used the balsam since March and seem to have completely overcome my trouble. — Yours truly.

Mrs. F. W. ROBESON.

WONDERFUL.

Baltimore, Md., Aug. 20, '24.

Dear Sir: — Please send me another bottle of this wonderful Aurine Ear Balsam. In enclose $2.00 for same. — Yours truly.

FRANK GORBI.

RECOMMENDS AURINE TO ALL

Johnstown, Pa., Aug. 21, 1924

Dear Sir: — I am enclosing two dollars for another bottle of Aurine Ear Balsam. I will say it has done wonders for me, for I have suffered with roaring in my ears for four years, and will recommend it to any one suffering with ear trouble. — Yours truly.

Mrs. LYDIA LANE.

ONE BOTTLE HELPED

Thurber, Tex., Aug. 6, 1924

Dear Sir: — Please send me by the next mail another bottle of your Aurine Ear medicine. I have used one bottle already and find it to be helping me. We will pay the postman as you said in your letter.

Sincerely yours,
Mr. CARL ROBERTS

Virex.—"Virex" is a widely advertised mail order fake put on the market by the Dale Laboratories of Kansas City, Mo. The same quacks that conduct the Dale Laboratories also have another piece of mail order quackery, called the Walker Institute, that sells an alleged cure for prostatitis. Virex was originally called "Rattlesnake Oil." There have been a number of socalled rattlesnake oils on the market, and none of them has ever been found to contain even a trace of snake oil. Not that it would mean anything if they had. One of the rattlesnake oils analyzed in the American Medical Asso-

Virex
COMPOUND
This Famous TEST
Proves the Penetrating Power
VIREX Quickly Reaches the Seat of Ear Troubles.
The Ear Is the Most Delicate and Intricate Mechanism in the Body
The Machine With Which We Hear
USE ONLY VIREX FOR EAR TROUBLES
THE DALE LABORATORIES, Kansas City, Mo.

ciation Laboratory was found to consist of oil of turpentine with small quantities of camphor, menthol and sassafras. (NOTE—On February 20, 1926, the Government declared Virex a fraud and debarred it from the U. S. mails.—A. J. C.)

MECHANICAL DEVICES.

Actina.—So much for the medicinal preparations. Of the mechanical devices there are a large number. Most of them are to be worn in the ears, and some are used in connection with medicaments. One, that some years ago had a very large sale, might be classed either with the medicinal preparations or with the mechanical devices. It was known as "Actina" and was described as a "perfect ozone battery" that would positively cure deafness and all diseases of the eye. A patent was granted on this device in 1886, demonstrating again the lack of discrimination used in the patent office in the issuance of patents on medicinal products and medical devices.

The device known as Actina was a metal cylinder with a large opening at one end and a small opening at the other. The large opening was applied to the eye to cure all diseases of that organ; the small end was applied to the nostrils to cure deafness. Within the device there was a small perforated cylin-

der that held absorbent material, soaked in a mixture of oil of mustard, oil of sassafras, amyl nitrate, hydrobromic ether and fluid extract of belladonna. The Actina had the odor of a decayed onion and could be counted on to make one weep copiously or sneeze violently, according to which end of the cylinder was applied. It was sold through the mails, and $10 was the price asked for it, although those who hesitated long enough had the chance of getting it for $5. For some years Actina brought in a fortune to its exploiters. Then the postal authorities declared the thing a fraud and debarred the exploiters from the use of the mails.

Deaf People Now Hear Whispers

Wireless Telephones for the Ears

They make the deaf hear distinctly.

They are out of sight when worn.

Wilson Ear Drums make sounds strike hard on the centre of the human ear drum, instead of spreading out weakly all over the surface.

Deafness from any cause, earache, buzzing noises in the head, raw and running ears, broken ear drums, and other ear troubles, are relieved and cured by these little wonder-workers.

A scientific book, about Deafness, tells how they are made, and has printed in it letters from hundreds of people who were deaf, but who now hear. Ministers, Lawyers, Physicians, Politicians, Governors, Soldiers, Workers in Factories, Farmers, tell how their hearing was restored, and how to obtain, almost instantly, the unique use of Wilson's Ear Drums.

This book has been the means of making 850,000 Deaf people hear again. It will be mailed free to you if you merely write a post card for it today. Get the free book for proof. You owe it to yourself and to your friends to learn about

Write for it to-day to the Wilson Ear Drum Co. 242 Tweed Building, Rochester, N.Y.

Wilson Ear Phone.—The ear drum type of deafness cures is a common one, and there are three or four that for some years have had a wide sale and are still extensively advertised. These are the Wilson Ear Drum, the Way Ear Drum, the Morley Invisible Ear Phone and the Mega Ear Phone. The Wilson device, according to the description

of its frenzied exploiter, is a wireless telephone for the ears. Wilson makes these modest claims for his device:

> "It is the only device in the world that collects sound waves and focuses them to one point."
> "It is the only device in the world constructed on scientific principles."
> "It is the only device in the world that is made to fit the orifice of the ear."
> "It is the only device in the world that cannot possibly injure the hearing."

The Wilson Ear Drum is generally sold on the mail order plan, although sometimes it is peddled over the country by its exploiter. It is a small rubber contrivance, made to slip into the external auditory canal, and sells for $5 a pair, with a nickel forceps and a drum inserter thrown in. Needless to say, the device has been granted a patent. More than ten years ago the Journal published a brief article on this piece of quackery, which in due course brought a letter from a lawyer, who stated that he represented Wilson. The attorney characterized the Journal's article as a scurrilous report that had caused his client irreparable injury and damage. He was asked to specify definitely any misstatements. Nothing further was heard from him. Some years later, however, Wilson assured a disgruntled purchaser of his wireless telephones that the American Medical Association has a fund of a quarter of a million dollars which it uses to put out of business anyone who tries to interfere with the practice of a physician who is a member of the American Medical Association. Thus are we advertised by our loving friends.

Way Ear Drum.—Another extensively exploited device is the ear drum—patented, of course—sold on the mail order plan by George P. Way of Detroit. We are asked to believe that these little rubber drums have enabled a man who was so deaf that he could not hear thunder to hear a clock tick thirty feet away. The distance of the thunder and the size of the clock are not specified. Way claims to have cured himself of twenty-five years' deafness by means of his drums. His sales method has been to offer the ear drums for $5 a pair, and to those who hesitate promise that if they purchase they will receive in addition a unique instrument which he has christened "Blowena," a "catarrh cure" side line. Way

calls his device a "medicated ear drum," as medicament is used in the device. In the older advertising Way claimed that he was the discoverer, inventor and patentee, although the specifications show that it is really the invention of Frances M. Way, his wife. She is the F. M. Way, described as the "well-known specialist" connected with the Way organization.

I Was Deaf 25 Years
I CAN NOW HEAR
with these soft, pliable artificial ear drums. I wear them in my ears night and day. They are perfectly comfortable. No one sees them in my ears. Write me and I will tell you a true story,—how I got deaf and how I made myself hear.
Medicated Ear Drum
Pat. Nov. 3, 1908
No. 902785
ARTIFICIAL EAR DRUM COMPANY
Address GEO. P. WAY, Manager
G. P. WAY
41 Adelaide Street
Detroit, Mich.

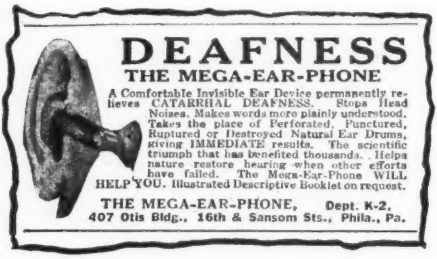
Morley Phone.—The Morley Invisible Ear Phone is one more extensively advertised device—also patented. It is put on the market by the Morley Company of Philadelphia. The thing is sold through the mails, and the Morley Company, in the past at least, has evidently disposed of the

letters it received from the deaf, as letter brokers have offered for sale and rent more than 17,000 Morley letters. Some of the claims made for the Morley Ear Phone are:

> "A miniature and invisible phone for the ear. Makes low sounds and whispers plainly heard."
>
> "The Morley Phone is the latest and most effective device for the relief of deafness."
>
> "It . . . transmits sound in increased volume to the auditory nerves.
>
> "It restores hearing like magic."

Unlike the rubber contrivances of Way and Wilson, the Morley Ear Phone is a small disc of oiled silk about one-quarter inch in diameter, through the center of which is a piece of silk thread. A piece of flexible tubing comes with the Morley device to aid in inserting it in the ear. The Morley Ear Phone, in other words, is nothing but the old Toynbee artificial drumhead, but made of oiled silk instead of rubber, as was the original.

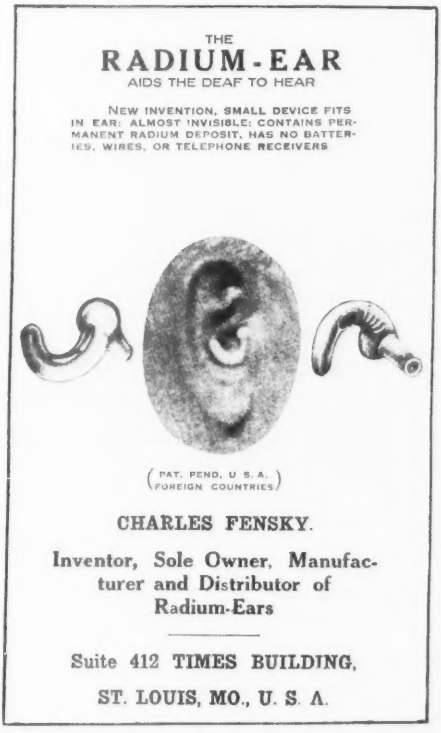
With a spool of silk thread and 10 cents' worth of oiled silk, anybody old enough to use a pair of scissors could make one hundred artificial ear drums that would be just as effective and just as potentially dangerous in the hands of the unskilled as the Morley phone. One hundred for 25 cents! The Morley phone sells for $5. Toynbee constructed his artificial membrane in 1852, and made it of rubber. Fifty years later the Morley concern applied for a patent on what is essentially the same thing, made of oiled silk, and the United States granted a patent on it as a new and useful improvement in artificial ear drums.


DEAFNESS
THE MEGA-EAR-PHONE
A Comfortable Invisible Ear Device permanently relieves CATARRHAL DEAFNESS. Stops Head Noises. Makes words more plainly understood. Takes the place of Perforated, Punctured, Ruptured or Destroyed Natural Ear Drums, giving IMMEDIATE results. The scientific triumph that has benefited thousands. Helps nature restore hearing when other efforts have failed. The Mega-Ear-Phone WILL HELP YOU. Illustrated Descriptive Booklet on request.
THE MEGA-EAR-PHONE, Dept. K-2,
407 Otis Bldg., 16th & Sansom Sts., Phila., Pa.

Mega Ear Phone.—Also from Philadelphia comes the Mega-Ear-Phone, on whose stationery appears the name of Edward Baum, M. D. This piece of fakery is sonorously described as an "invisible, multiple, sound accumulating, megaphonic augmentation and cornupial accumulator." It is, we are told, the "latest and most effective device for the relief of impaired hearing of any degree." The patent was granted in 1916. The Mega-Ear-Phone outfit consists of six pairs of Mega-Ear-Phones, made of oiled silk, a small hard rubber tube, split at one end, to be used in insert-

ing the device, a small bottle of oil, a rubber ear syringe, a few wooden applicators, a small wad of cotton and a pair of tweezers. The outfit sells for $10.

The Audiphone.—From St. Louis comes the Audiphone, put out by one Charles Fensky. This is a metal device,


THE
RADIUM-EAR
AIDS THE DEAF TO HEAR
NEW INVENTION, SMALL DEVICE FITS IN EAR; ALMOST INVISIBLE; CONTAINS PERMANENT RADIUM DEPOSIT, HAS NO BATTERIES, WIRES, OR TELEPHONE RECEIVERS
(PAT. PEND. U. S. A. FOREIGN COUNTRIES)
CHARLES FENSKY.
Inventor, Sole Owner, Manufacturer and Distributor of Radium-Ears
Suite 412 TIMES BUILDING,
ST. LOUIS, MO., U. S. A.

having a straight cylinder to fit the external auditory canal, and a curved portion that hugs the concha. The chief element of hokum in the Audiphone is not, however, its mechanical structure, but the claim that the device is radioactive. Originally it was said to be charged with "Hearium," described as a radioactive substance discovered by one Abbott E. Kay of

Chicago and Glen Ellyn. Two or three years ago Kay was dealing in fake radium. He was distributing to physicians what he described as a plaque containing 25 milligrams of official radium—whatever that is. The Bureau of Investigation collected some of these plaques and submitted them to the Bureau of Standards at Washington, which also tested other specimens sent in from independent sources. In every instance the product was found to be without any radioactivity. Then the Federal Trades Commission got after Kay and ordered him to cease selling or offering for sale or advertising as radium the product previously sold by him as radium. He was also ordered to cease employing the word radium in connection with the product that he had been selling.

The next we heard of Kay was in connection with Fensky's Audiphone. A physician was asked to write to Kay for information about this product "Hearium," the mystery element of Fensky's device. Kay replied that "Hearium is Kay's 436-3, a radioactive derivative of a group of minerals authentically stated to abound in vanadium." Elaborating further, Kay went on to state that "Hearium is radium of Kay extraction, plus an admixture which experience shows modifies the effects on the ear." In the same letter Kay declared that with "Hearium" he had cured a case of deafness in forty-eight hours in a patient who had been deaf for thirteen years.

Later Fensky declared that he had abandoned "Hearium" as an ingredient of the Audiphone and was now using an actual deposit of radium in extremely low dosage, prepared by Dr. T. J. Randall of Los Angeles. Dr. Randall has long specialized in autohemic therapy, orificial surgery and intravenous medication, and when the Abrams fake came along he opened the Glendale Electronic Institute. Now it appears he is charging Fensky's Audiphones with low dosage radium.

The physician who had previously written to Abbott E. Kay, wrote again telling Kay that Fensky now claimed to have abandoned the use of "Hearium," because it did not prove satisfactory. Kay countered with the statement that the reason for abandoning "Hearium" was that Fensky owed him over

$6,000 for previous services. Kay closed his letter with the suggestion that the physician to whom he was writing might care to take up the remunerative field that Fensky was relinquishing.

Hearing Restored
by the AUDIO Treatment
Our Patients
Furnish the Proof
This is not an "aid to hearing," nor an appliance to be worn in the ear, or on the person. It is a treatment administering a sound-wave vibratory massage so perfectly controlled that persons in all stages of deafness can be made to instantly hear both voice and music.
If the arm is broken and carried in a sling for weeks, when the sling is removed, the arm is useless. Its usefulness is restored by massage and exercise. In the treatment of impaired hearing, we apply the same principle to the ear, introducing a sound-wave vibratory massage and exercise which induce normal hearing.
If the delicate bones and muscles of the middle ear have become rigid, or the functioning of the ear is interfered with, this sound-wave vibratory massage tends to loosen the rigidity, to strengthen the muscles, and induce proper blood circulation to return to these parts, aiding nature in the restoration of a healthy and functioning condition.
If you are deaf, hard of hearing, or troubled with head noises, call and let us give you a private demonstration of the Audio which makes possible the restoration of your hearing to a state of practical usefulness.
AUDIO INSTITUTE, INC.
Suite 1612 Mallers Building, 5 S. Wabash Ave., Chicago
Phone Central 2422. Office Hours: 9 A. M. to 6 P. M. Daily, Except Sunday
Free Demonstration

The Deafone.—This paper would be incomplete if it failed to refer to a device that is being exploited locally as a cure for deafness. It was originally called the "De-a-fone," and was put on the market by a concern that was first known as the National Acoustic Laboratories. Later it became the National Deafone Company, and within the past few weeks has been changed to Electrophone Corporation. The instrument, which is now rechristened "Electrophone," is being used

in the socalled Audio Institute, which advertises in the Chicago papers "Hearing restored by the Audio treatment."

At one stage of the exploitation of the Electrophone it was claimed that the device had been approved by three well known Chicago aurists. The facts were not one of the three would give his approval. On the contrary, they expressed opinions that it was without value. One preposterous claim that has been made for this device is that "with the De-a-fone any physician can today accomplish far more mechanically than even the most optimistic aurist ever hoped to accomplish by any other means." Further, we were told that "with the De-a-fone we go directly to the seat of the affliction and with proper technique satisfactory results are assured."

An interesting sidelight on the sales methods of the concern appears in a letter sent by the National Deafone Company to a layman who lives in a Missouri village of less than two thousand population. This layman was told that De-a-fone machines were sold to institutions, hospitals, sanitariums and physicians, and the company suggested that he make an effort to interest some physician who was financially able to invest $2,500 for one of the machines, and then arrange to take daily treatments. The National De-a-fone Company assured this man: "We are positively restoring hearing every day." They also declared that nationally known otologists and physicians who had examined the De-a-fone had, without exception, expressed the opinion that it was the most "wonderful constructive scientific apparatus ever given to humanity." With the letter to the layman there was a small advertising leaflet in which it was stated, among other things, "We have yet to find a single deafmute who cannot be made to hear by our method." Just what value, if any, this device has may still be a moot point, but the fact that an aurist of unquestioned ability who used it in an experimental way rejected it on the grounds that the results did not warrant further investigation, would indicate that the De-a-fone, or, as it is now called, "Electrophone," has no large field of usefulness.

PRACTITIONERS.

Guy Clifford Powell and L. C. Grains.—Now to deal for a few minutes with a few practitioners in the field of

aural quackery and pseudomedicine. The late Guy Clifford Powell of Peoria immediately comes to mind as one of the best known of the aural quacks. Powell called himself an international specialist, and he was, in fact, an international quack, for he advertised on both sides of the Atlantic. His particular piece of buncombe was the "Electro-Vibratory Cure for Deafness." He did business on the mail order plan, asking $100 in his first letter, but—sliding swiftly through a series of form letters—coming down finally to $15.

In due time Powell was gathered to his fathers. His business, too valuable an asset to let die, was for a while continued by the Dr. L. C. Grains Company, which was practically owned by an advertising agency—the Charles H. Fuller Company of this city. L. C. Grains himself seems originally to have had a small mail order fakery of his own, specializing in "Red Clover Extract" and "Curo Grains of Life." After taking over the Powell factory and moving it from Peoria to Chicago, the L. C. Grains Company was unable to carry on, and the concern lasped into innocuous desuetude.

Dr. Edward E. Gardner.—This individual, from New York City, has described himself as a "celebrated New York aurist." Many years ago Gardner practiced in Illinois, but sought richer fields in the east. For a while he was with the Copeland Medical Institute of New York City. Later he and his father-in-law exploited a catarrh cure under the name

of the Norma Medical Association. Still later he organized his deafness cure in connection with his brother, who is not a doctor, and conducted the business under the style of "Drs. Gardner" until official intervention caused a change in title. Incidentally Gardner was a stockholder in the "Help-to-Hear Company," a swindle that the federal authorities put out of business in 1906. Gardner went into bankruptcy in 1907 with about $13,000 liabilities and no available assets. The Norwich Pharmacal Company was said to have been one of his largest creditors. Gardner's methods were typical of the mail order quack: Form letters, free trial treatment and a sliding scale of prices from $5 to $1.50. He also had some side lines, including a rheumatism treatment for $1, insomnia also came at $1, as did hysteria, indigestion and blackheads, while he would cure constipation for 50 cents.

G. M. Branaman.—For many years G. M. Branaman of Kansas City, Mo., conducted a deafness cure mail order concern. Then the federal authorities got around to him, declared his business a fraud and debarred him from the use of the United States mail.

Branaman had a somewhat elaborate treatment. There was what he called an "electro-magnetic head-cap," which was to be worn by the patient. This was said to produce a current of electricity which, it was alleged, would revivify the nerves and other structures of the ear. There were also some tablets to be taken before each meal, some ear drops to be used each night, a vaporizer liquid and a liquid for a gargle. The tablets were strychnin sulphate, 1/60th of a grain. The ear drops contained about 3 per cent carbolic acid, 25 per cent glycerin and 72 per cent saturated solution of boric acid. The vaporizer liquid was more than 97 per cent liquid petrolatum with small quantities of menthol, camphor and Thuja oil. The gargle was mostly water, with a little glycerin, bicarbonate of soda, borax and a dash of carbolic acid.

The government in its case against this quack proved that he had intentionally accepted cases of incurable deafness for treatment and promised a sure cure. It proved, further, that Branaman published a testimonial to the effect that one John Turner's hearing was perfect and that he had been cured by

SUCCESSFULLY TREATED
he tissues waste, stomach and
rform their natural function
on becomes impaired, the kid
s composed of two great sy
erebro-spinal system, comp
n and spinal cord and the ne
connected with the centers wh
nimal life, and the organic syste
of nerves controlling the or
other words, the actio
the condition
, the nerves unstrung and mucous
discharges, gradually destroyi
, smell, hearing and memory. M
reaches the different parts of
supplying pure medicate
al passages, bronch

Branaman. The fact was, and Branaman knew it, that Turner was so deaf that he was incapacitated for work. Branaman was a persistent faker. After the fraud order had been issued he applied for an injunction against the postmaster of Kansas City to restrain the enforcement of the order. The injunction was denied by the court, which in reviewing the case held that the evidence amply sustained the issuance of the fraud order. Branaman's plea was that the action of the Postmaster General was the result of a conspiracy on the part of the American Medical Association and government officials.

George E. Coutant, M. D.—This man, another quack from New York City, cut a wide swath a few years ago in advertising a mail order cure for deafness. When salvarsan was first introduced in this country Coutant, under the trade style "'606' Laboratories," advertised "One Dose Cures, All Symptoms Removed in Two Days." Even before that, Coutant had been involved in other forms of quackery equally disreputable. In the first of his long series of follow-up letters on his deafness cure scheme he asked $10 for the treatment, which was

reduced by easy stages until in the seventh it was offered for $3. The treatment itself consisted of several small boxes of gaily colored pills and tablets, a salve, a nickel plated inhaler and a nasal douche.

Coutant's trump card was his testimonials. We took the trouble to investigate some of these. Several of the testimonial givers were written to. Eight of them replied. Seven of these repudiated their testimonials. The one that stood up for Coutant we found on investigation was in the quack's pay.

Coutant, as is common practice with most mail order quacks, used what he called a "Money Refund Guarantee Certificate," which a careless reading led one to believe that in case the patient were not benefited Coutant would return the money. What the refund certificate really did promise was that if three complete treatments were purchased at $10 each, the price of the first treatment would be refunded if the patient declared that he had not been benefited. In other words, Coutant played to get at least $20 for something not worth 20 cents. Yet most of his victims when they first ordered, believed that Coutant guaranteed either to cure them or to refund their money.

W. O. Coffee.—From Davenport, Ia., Dr. W. O. Coffee conducts his mail order deafness cure. Coffee used to do an extensive bunko business from Des Moines in curing eyes by mail. In those days part of his advertising paraphernalia were some lurid reproductions in color from Haab's "External Diseases of the Eye." One of the Haab illustrations, which in the original was labeled "Lime-burn, caused by the explosion of a

WANTED! 15,000 SUFFERERS FROM
DEAFNESS
HEAD NOISES
Or Nasal Catarrh
I want the names of 15,000 people who are suffering from cartarrhal deafness, dullness of hearing or head noises. I have an original home treatment for these distressing conditions which I want you to try without cost or expense. I suffered for many years with catarrh, deafness and head noises, tried many different treatments. Had two surgical operations all of which failed to give permanent relief. I began experimenting on myself and finally found a treatment which completely healed my catarrh, restored my hearing and stopped the terrible head noises. I want you to try this splendid treatment entirely at my own expense.
DR. W. O. COFFEE, Noted Catarrh and Deafness Specialist
15,000 TREATMENTS TO BE GIVEN THIS MONTH FREE!
I am so proud of my treatment and so anxious to help others suffering with deafness, head noises or catarrh that I will have packed and ready for shipment 15,000 demonstration treatments which I will give away in the next 30 days absolutely free. I want to prove that my original home treatments will give relief from catarrhal deafness, head noises or nasal catarrh. The results are quick and convincing. You feel the difference the first day. This treatment costs you nothing whatever. Simply write and say that you are suffering with deafness or head noises or catarrh I will gladly send the free treatment. It is the best treatment I have ever found in over 40 years daily practice as eye, ear, nose and throat specialist. I want every sufferer with deafness, dullness of hearing, head noises or catarrh to try it free.
Don't Neglect Deafness
Catarrhal deafness, head noises and catarrh when neglected gradually grow worse. Authorities claim that 90% of all deafness is caused primarily by catarrh of the head and air passages. Others state that catarrh causes indigestion; rheumatism, catarrh of the stomach and many other diseased conditions.
Try It Free
I don't ask you to send one penny for this treatment. I send it free to you prepaid without cost. I am doing this to quickly introduce my original home treatment to sufferers of catarrhal deafness, head noises or catarrh. I believe I treat more cases of deafness, head noises and catarrh than any physician in the United States. I know this treatment is the best I have ever used. Send your name and address today. State whether you have deafness or head noises or just have catarrh. This notice may not appear again so write me at once.
DR. W. O. COFFEE, Suite 585 St. James Hotel Bldg., Davenport, Iowa

bottle," was reproduced by Coffee and carried this legend: "This eye was afflicted with granulated lids and ulcers, following inflammation. There is no known remedy that will remove these spots except Dr. Coffee's absorption treatment, and it will do it completely." Another illustration from Haab's book showed a lime-burn of longer standing. This, under Coffee's interpretation, became "Chronic ulcers of the eye and cataract." Still another plate in Haab's book showed two specimens: one, senile cataract in a women 72 years old, and the other cataract in a 14-year-old boy. This plate was appropriat-

ed by Coffee and became before-and-after exhibit. The patient, said Coffee, made the fatal error of submitting to an operation with the result shown in "A," but afterwards came to Coffee, who repaired the damage as shown in "B."

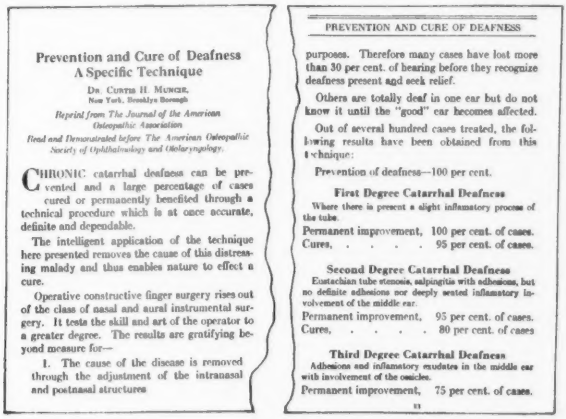
Samuel Hopkins Adams, in his castigation of quacks in Collier's, devoted some space to Coffee and his methods. After this unfavorable publicity Coffee's mail order eye business seemed to wane, and in 1915 the Des Moines newspapers recorded the fact that Coffee had gone into voluntary bankruptcy. His heaviest creditor, according to the report, was Arthur Capper, at that time Governor of and now Senator from Kansas, and a publisher of papers that have long been known for their easy virtue in the matter of quack advertising.

His mail order eye cure failing, Coffee turned his talents to the itinerant field and moved from Des Moines to Davenport. For a while he was especially partial to Illinois, which he apparently found a lush field. Gradually, from curing eyes, he switched over into the deafness cure field. Still more recently he has abandoned itinerant aural quackery and has gone back to the easier and possibly equally lucrative branch of mail order quackery, still, however, as an aurist.

Curtis H. Muncie, Osteopath.—This paper would not be complete without at least a passing reference to Curtis H. Muncie, the Brooklyn osteopath, who claims to be curing deafness by means of what he calls "operative constructive finger surgery." More than five years ago a newspaper service that furnishes sensational features for certain Sunday newspapers, dished up a full page article dealing with the alleged cure of the congenital deafness of young Prince Don Jaime, second son of the King of Spain. The story was to the effect that a London bone setter, by finger manipulation, had corrected a displacement alleged to be the cause of the Prince's deafness. Investigation showed, of course, that the story was a pure fabrication. It was too good to waste, however, and soon the osteopaths began reproducing it, modifying it in such a way as to credit osteopathy with the alleged cure; later the chiropractors used the same material, with a member of that cult playing the stellar part.

Then, in the summer of 1923, papers in various parts of the country carried a story to the effect that Curtis H. Muncie, a

Brooklyn osteopath, had been called to Europe to treat Prince Don Jaime for his deafness. A New York paper reported that Muncie by his manipulations had cured the Prince of his ailment and had declared that the lad's deafness was due to "a state of complete deformity" of the eustachian tubes. Muncie's treatment was simply that of "reconstructing the eustachian tubes"—a mere bagatelle for an osteopath. The yarn was a readable one, and its advertising value to Muncie must have been worth a king's ransom. Investigation proved that Prince Don Jaime was still deaf and had undergone no such treatment as that reported.



Prevention and Cure of Deafness
A Specific Technique

DR. CURTIS H. MUNCIE,
New York, Brooklyn Borough

Reprint from The Journal of the American Osteopathic Association
Read and Demonstrated before The American Osteopathic Society of Ophthalmology and Otolaryngology.

CHRONIC catarrhal deafness can be prevented and a large percentage of cases cured or permanently benefited through a technical procedure which is at once accurate, definite and dependable.

The intelligent application of the technique here presented removes the cause of this distressing malady and thus enables nature to effect a cure.

Operative constructive finger surgery rises out of the class of nasal and aural instrumental surgery. It tests the skill and art of the operator to a greater degree. The results are gratifying beyond measure for—

1. The cause of the disease is removed through the adjustment of the intranasal and postnasal structures

PREVENTION AND CURE OF DEAFNESS

purposes. Therefore many cases have lost more than 30 per cent. of hearing before they recognize deafness present and seek relief.

Others are totally deaf in one ear but do not know it until the "good" ear becomes affected.

Out of several hundred cases treated, the following results have been obtained from this technique:

Prevention of deafness—100 per cent.

First Degree Catarrhal Deafness
Where there is present a slight inflamatory process of the tube.
Permanent improvement, 100 per cent. of cases.
Cures, ... 95 per cent. of cases.

Second Degree Catarrhal Deafness
Eustachian tube stenosis, salpingitis with adhesions, but no definite adhesions nor deeply seated inflamatory involvement of the middle ear.
Permanent improvement, 95 per cent. of cases.
Cures, ... 80 per cent. of cases

Third Degree Catarrhal Deafness
Adhesions and inflamatory exudates in the middle ear with involvement of the ossicles.
Permanent improvement, 75 per cent. of cases.

11

On Muncie's return from Europe, he announced that the earlier report was not altogether accurate; that while it was perfectly true that he had gone to Europe to operate on a prince, it was not Prince Don Jaime, and he would not divulge who it was. The newspaper carrying this correction quoted Muncie as having said that after a twenty minute operation the prince on whom he did operate had practically normal hearing. According to Muncie's advertising booklet, he obtains the following results from his methods of treating deafness: In the prevention of deafness, 100 per cent success. In what he calls first degree catarrhal deafness, he produces permanent improvement in

100 per cent and cures in 95 per cent. In second degree catarrhal deafness he produces permanent improvement in 95 per cent and cures in 80 per cent. In cases of deafness in which there are adhesions and inflammatory exudates in the middle ear, with involvement of the ossicles, he gets improvement in 75 per cent. The same booklet describes his technic. With the patient under nitrous oxid anesthesia he "normalizes and reconstructs" the eustachain tubes, "adjusts" the nasal tissues, turbinates and septa, and with his trained finger "feels adenoids, pus-pockets and adhesions and removes them." Then the patient "comes back from a pleasant sleep with a reconstructed nasal and nasopharyngeal area." His fees, according to a letter that we have on file, are: Examination, $25; operation, $200 and up, with postoperative treatments, two a week for six months, at $10 each.

The time allotted is up, and yet the surface has been barely scratched. The scope of aural quackery is so broad, and the number of fakes and fakers, fads and faddists so great that a book might be written on the subject. Aural pseudomedicine will continue to flourish just so long as public credulity obtains, for, to paraphrase Voltaire, the quack was born when the first knave met the first ignoramus. It is lack of knowledge and not a dearth of brains that breeds credulity. The best that the medical profession can do in protecting the public is to turn the light on the methods of the quack, so that his ignorance and sordidness become apparent. In this way the layman can acquire a certain degree of critical judgment that will make him a less easy prey for the medical shark. The quack's philosophy was well expressed by Talleyrand: "To succeed in the world it is much more necessary to possess the penetration to discover who is the ignoramus than to discover who is the wise man."

VII.

THE INFLUENCE OF PARTIAL AND COMPLETE OCCLUSION OF THE EXTERNAL AUDITORY CANALS ON AIR AND BONE TRANSMITTED SOUND.

By A. G. Pohlman, M. D.,

DEPARTMENT OF ANATOMY, ST. LOUIS UNIVERSITY,

AND

F. W. Kranz, Ph. D.,

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The experimental evidence submitted in our paper published in the preceding issue of this journal established, first, that both bone and air transmitted sounds normally enter the cochlea through the stapedial footplate; second, that the acuity for air transmitted sound is not in any detailed sense a criterion for the acuity of bone transmitted sound; and third, that the advantage to bone transmission acuity through occlusion of the external auditory canals is dependent upon the frequency of the tone exhibited and is extremely variable between individuals. It was pointed out that one individual (B) was appreciably more sensitive for bone transmitted sound than were the other three individuals reported on. This same individual also showed the least advantage on occlusion of the external auditory canals. It is, of course, well known and was first pointed out by Rinne that partial occlusion of the external auditory canals also enhances the acuity for bone transmitted sounds. It was thought desirable to determine quantitatively the effect of partial occlusion on bone acuity and to determine whether an explanation might be made of the unusual bone sensitivity of subject B on the basis of a natural partial occlusion. The experiment was divided into two parts; first, a determination of the effect of complete occlusion on air acuity, and second, the effect of partial and complete occlusion on bone acuity.

Five individuals were tested for air acuity for five frequencies: 128, 256, 512, 1024 and 2048 p. p. s. The air acuity was determined by the use of a calibrated telephone receiver provided with a rubber cushion to avoid direct pressure on the occluded canals. The rubber cushion necessarily influences the amount of activation of the receiver required for minimum audition as compared with that required with the receiver applied directly to the head because of the greater volume of trapped air. We were, however, interested only in the differences, and the conditions for testing with and without occlusion were the same except perhaps for the plug in the canal itself. Each ear was tested separately. With the electrical current at a fixed value, the number of attenuation units* introduced into the electrical circuit to bring the tone to minimum audition was noted, first without any interference to the hearing and next with the external auditory canal occluded with dental impression wax.

Thus in the right ear of subject I, 3.2 units were required for minimum audition with the ears open and 1.2 units with the ears occluded, a difference of 2 units, or a ratio of 100 in intensity. This means that it takes 100 times the intensity for subject I to hear the pitch 128 p. p. s. with the ear occluded than it does with the ear open. The technic for the test is described in greater detail in our preceding article on bone conduction. Occlusion with the finger is extremely unsatisfactory, not only because of the variability in amount of occlusion and of pressure in the external auditory canal, but also because the finger introduces adventitious noises. Then, too, it is, of course, quite impossible to test with a telephone receiver an ear stopped with a finger tip. The ears were occluded by massaging a small amount of warm dental impression compound into the external auditory canal. This gives

*The units of attenuation used in this paper are those used and discussed in a previous paper by one of the present authors (Kranz, "Minimum Intensity for Audition," Physical Review, V. 21, p. 579, May, 1923). They are defined by $n = \text{logarithm of } m$, or $m = 10^n$, where n is the number of attenuation units corresponding to an energy ratio of m. Thus one attenuation unit corresponds to an energy ratio of 10, two units to a ratio of 100, four units to a ratio of 10,000, etc., while one-half unit corresponds to an energy ratio of $\sqrt{10}$ or 3.2. This is the simplest unit of the kind which can be devised and the relation is easy to remember.

a perfect seal without discomfort, except perhaps in the removal of the hardened material.

The information on the five individuals is given in Table 1. Data on the right and left ears of each individual are shown separately at each of the five frequencies used, separated into three groups. The first group indicates the relative sensitivity of each unobstructed ear in attenuation units; the second group shows similar data with the ear occluded; and the third group gives the differences between the the first two and so represents the effect of the occlusion. The figures of this third group were averaged, and the resulting number of attenuation units were transformed into relative intensities or the actual number of times the unobstructed minimum intensities had to be increased in order to hear through the plug of material. The minimum audition for these five subjects is practically normal (in comparison with average data on a large group), except perhaps for a slight deficiency of subject IV at 256 p. p. s. and subject V at 2048. It may also be noted that the size of the plugs varied somewhat because of the difference in the various external canals, and this may have had a slight influence on individual variations in addition to the occlusion.

Summarizing the information, we find that to hear the pitch 128 p. p. s. through the plug requires, on the average, 14.5 times the intensity needed without the plug in the external auditory canal. At 256 p. p. s. an increase of 40 times is required; 190 times at 512; 331 times at 1024; and 10,000 times at 2048 p. p. s. The occlusion of the external auditory canal therefore does not effect the same degree of blocking against all frequencies. This is an important point and has considerable practical significance.

During the late war an attempt was made to protect the soldiers against the noise and concussion without making them deaf to spoken commands, by occlusion of the external auditory canals. The experiment would have been more successful if the requirements had been reversed, because an individual with occluded canals is more deaf to voice sounds than he is to the noises about him, due to the differential effect of the occlusion, because speech involves higher frequencies than do general noises. Subjectively we sometimes try to gain an idea of what a person with conduction deafness hears by placing

our fingers in our ears. However, in doing this we lower our acuity for high pitched sounds more than we do for those of low pitch, while a conduction deaf individual has lowered acuity for low pitched sounds and is not so deaf for the higher frequencies. It is quite impossible for a normal hearing person to imitate the condition of conduction deafness by blocking the external auditory canals.

The occlusion of the external auditory canals, even when done as thoroughly as in these tests, will not materially protect the individual from auditory insult in an occupation where he is constantly exposed to low pitched noises. It would be much better to select for such occupations individuals who are of the conduction deaf type, because they will not only be less troubled with the noise but will also be able to understand a normal hearing foreman all the better. A foreman with normal hearing will subconsciously increase the intensity of his voice in the presence of a noise, and will therefore be more easily understood by conduction deaf individuals who are not so pronouncedly affected by the noise. The problem of paracousis Willisi is considered in another paper.[1]

We considered the possibility that the introduction of the rather large wax plug into the cavity enclosed between the telephone receiver and the head might have brought in factors other than the particular effect which we wished to test. Therefore, the tests were repeated with a telephone receiver serving as a source of sound and lying on a soft pad on the table at a distance of about a foot in front of the observer. The tests were made in a room padded with felt over all of its surface and so reflections did not have to be considered. In this case the ears were not tested separately, but both ears of an individual were plugged at once and an observation was made on his acuity in the occluded condition and again with the ears open in the normal condition. The results obtained were very similar to those detailed above and verified the conclusions drawn as to the magnitude of the effect and the variation with frequency.

The second experiment was performed on eight individuals, five of whom were the five of the preceding test. The bone activating receiver was placed on the forehead and, with a given initial electrical current, the number of attenuation units

required for reduction to minimum audition was established for the following three conditions: 1, with ears open; 2, with external auditory canals partially occluded; 3, with the canals completely occluded. Partial occlusion was obtained by sealing a glass tube 2.5 cms. long and of 4 mm. lumen into each canal with warm dental impression compound. Plugging the ends of the tubes with plastocene resulted in complete occlusion. The results are submitted in Table 2 in a manner similar to that used in Table 1. The first group shows the number of attenuation units necessary to reduce a fixed initial intensity down to minimum audition with the ears open, so that the relative normal sensitivities to bone conduction may be noted for the different individuals. The second group indicates in attenuation units the amount of improvement brought about by partial occlusion. The third group shows the additional improvement which complete plugging of the canals produced over the partial occlusion. The fourth group shows the number of units of improvement in complete occlusion over the sensitivity with the ears open, and so is the sum of the second and third groups. The averages for each of the three conditions are given in attenuation units, and also transformed into actual intensity ratios.

The averages of the eight cases are shown in Fig. 1 in graphic form, the intensity ratios of improvement being given for each of the five frequencies used. A logarithmic scale is used for the intensity ratios, as this gives a truer idea of their relative importance. Ratios greater than 1 indicate that the bone acuity was improved by increase in occlusion, while the one point below the base line 1 means that the average effect of the indicated increased occlusion was detrimental to bone acuity.

It will be seen that the effect of partial occlusion is not large at 128 p. p. s., but the advantage becomes increasingly prominent up to 512, drops off slightly at 1024 and has no effect at 2048 p. p. s. The advantage of complete occlusion over partial occlusion is most pronounced at 128 and gradually drops off to a negligible effect at 1024 and 2048 p. p. s. Complete occlusion shows its advantage over the unoccluded condition more at the lower frequencies than at the higher, rising somewhat from 128 to 512, decreasing at 1024 and be-

coming negligible at 2048 p. p. s. The enhancement to bone transmission at 1024 p. p. s. is almost entirely due to partial occlusion. The lines connecting the experimental points do not imply any knowledge of conditions at frequencies other than those tested.

It is probably impossible to place all the ears under exactly the same conditions of occlusion—that is, with the wax in contact with the same area of the surface of the canal, and it is possible that this may have played some small part in the variation between individuals shown in the results, but the major portion of the variation is undoubtedly due to true variation between individuals.

Translated into clinical experience, a small amount of cerumen will have a greater lateralization effect at 512 p. p. s. than at 256 or 1024, on the average. However, in the Weber-Wheatstone tests a fork of 256 p. p. s. will be more satisfactory than those usually employed. At 108 or 128 p. p. s. there is danger of confusing tactile sensation with hearing, and at 512 the patient may hear the air sound in the other ear. Disregarding subject I, the other seven observers grouped more closely at 256 p. p. s. than at any other frequency, for bone conduction with open ears.

Subject I of this series showed, as subject B of the preceding paper, a marked acuity for bone transmitted sounds at 256 p. p. s., and the suggestion had come to mind that this individual might normally be in a condition comparable to partial occlusion in the other subjects. However, some of the individuals used in the present tests, but not in those of the preceding paper, showed a similar high acuity with ears open and small gain on occlusion at some frequencies, for instance, subjects IV, VI and VII at 128 p. p. s. So subject I is not wholly unique, and the hypothesis of a normal condition of partial occlusion of subject I hardly seems to be justified, although his superiority in bone acuity at 256 is outstanding. He must be regarded as a subject with prolonged bone transmission or greater sensitivity than the average at 256 p. p. s., but no mechanical explanation is offered to account for this condition. In general, the higher bone acuities seemed to result in the lesser gains on partial occlusion, although in some cases low gains on partial occlusion were associated with low initial acuities.

There are several explanations offered for the Weber-Wheatstone phenomenon which will be discussed in a later and more complete paper on lateralization. The evidence here submitted supports the original contention that the advantage to bone acuity on occlusion of the external auditory canal is definitely related to the frequency, and the information on the relation of partial occlusion to the normal condition and to complete occlusion is quite new. In any event, any mechanical factor suggested as the cause of the Weber-Wheatstone phenomenon must consider not only the complete occlusion condition but also that of partial occlusion and the differences between these two in the various parts of the frequency range.

TABLE I.

EFFECT ON AIR ACUITY OF OCCLUSION OF EARS.
FIVE SUBJECTS.

Ears Not Occluded

Frequency	I		II		III		IV		V		Average	Average Detriment given as Intensity Ratio
	R	L	R	L	R	L	R	L	R	L		
128	3.2	3.6	3.6	3.2	3.2	3.2	2.8	2.8	3.2	3.2		
256	6.4	6.4	6.0	6.0	6.0	6.0	5.2	4.0	6.8	7.2		
512	7.2	7.2	7.6	7.2	7.6	7.6	6.4	7.2	7.6	7.6		
1024	7.6	7.6	7.6	7.2	7.6	8.0	7.6	6.4	6.8	6.8		
2048	5.6	5.6	5.2	5.2	5.6	5.6	5.6	5.6	3.2	4.4		
Ears Occluded												
128	1.2	1.2	2.4	2.4	2.8	2.4	1.6	1.6	2.0	2.8		
256	4.4	4.4	4.0	4.8	4.4	4.0	4.4	3.2	5.2	5.2		
512	4.0	4.0	5.2	5.2	6.0	5.2	4.0	4.0	6.8	6.0		
1024	3.6	4.0	4.8	4.4	5.2	5.6	5.2	4.8	5.6	4.8		
2048	0.8	0.8	1.2	1.6	2.0	2.4	1.2	2.0	-0.4	0.		
Difference Due to Occlusion												
128	2.0	2.4	1.2	0.8	0.4	0.8	1.2	1.2	1.2	0.4	1.16	14.5
256	2.0	2.0	2.0	1.2	1.6	2.0	0.8	0.8	1.6	2.0	1.6	40.
512	3.2	3.2	2.4	2.0	1.6	2.4	2.4	3.2	0.8	1.6	2.28	190.
1024	4.0	3.6	2.8	2.8	2.4	2.4	2.4	1.6	1.2	2.0	2.52	331.
2048	4.8	4.8	4.0	3.6	3.6	3.2	4.4	3.6	3.6	4.4	4.0	10,000.

TABLE II.

EFFECT ON BONE ACUITY OF PARTIAL AND TOTAL OCCLUSION.
EIGHT SUBJECTS.

Ears Not Occluded

Frequency	I	II	III	IV	V	VI	VII	VIII	Average	Average Improvement given as Intensity Ratio
128	1.4	-.2	0.	1.2	0.4	2.0	1.2	0.		
256	2.8	1.2	0.8	0.4	1.2	0.8	1.2	1.2		
512	1.8	1.4	1.2	0.	0.8	0.8	2.0	0.4		
1024	3.0	3.2	4.0	1.6	1.6	2.0	2.4	2.4		
2048	1.9	1.3	1.2	2.4	0.	2.0	1.6	2.0		
Advantage of Partial Over No Occlusion										
128	0.3	1.4	0.8	0.4	-0.4	0.	0.4	1.2	0.5	3.2
256	0.4	1.8	1.2	0.	0.4	2.0	1.6	0.8	1.02	10.5
512	1.0	1.6	1.6	1.6	1.2	1.6	0.8	2.4	1.48	30.2
1024	1.0	1.4	1.2	1.2	1.2	1.6	2.0	0.8	1.3	20.
2048	1.2	-1.3	0.4	0.	0.4	0.	0.4	-1.2	0.	1.
Advantage of Total Over Partial Occlusion										
128	1.4	1.4	2.0	0.4	1.2	1.6	1.6	1.6	1.4	25.
256	0.6	0.8	1.6	1.6	0.8	1.2	0.8	1.2	1.08	12.
512	0.6	0.2	0.8	1.2	1.2	1.6	0.8	0.4	0.84	6.9
1024	0.4	-0.2	-0.4	0.4	0.	0.	0.	-0.8	-0.08	0.83
2048	0.2	0.6	0.	0.4	0.	0.4	0.	0.	0.2	1.6
Advantage of Total Over No Occlusion										
128	1.7	2.8	2.8	0.8	0.8	1.6	2.0	2.8	1.9	79.
256	1.0	2.6	2.8	1.6	1.2	3.2	2.4	2.0	2.1	126.
512	1.6	1.8	2.4	2.8	2.4	3.2	1.6	2.8	2.32	2.09
1024	0.4	1.2	0.8	1.6	1.2	1.6	2.0	0.	1.22	16.6
2048	1.4	-0.7	0.4	0.4	0.4	0.4	0.4	-1.2	0.2	1.6

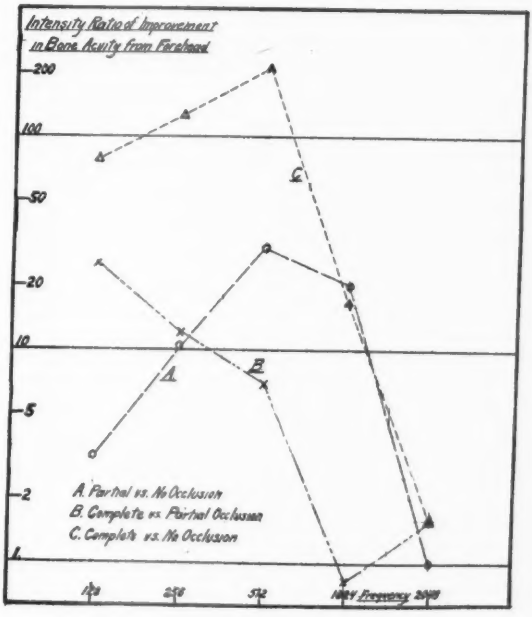
Intensity Ratio of Improvement
in Bone Acuity from Forehead
200
100
50
20
10
5
2
1
A
B
C
A. Partial vs. No Occlusion
B. Complete vs. Partial Occlusion
C. Complete vs. No Occlusion
128
256
512
1024 Frequency 2048

Fig. 1.

VIII.

AUDITORY FACTS AND THEORIES—(CONTINUED).* THEORIES OF AUDITION.

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LOS ANGELES.

The problem "how we hear" has intrigued philosophers and scientists throughout the ages. The early Greeks considered that as the sound waves are communicated through the air, the internal ear also probably contained this same medium. Later, it was discovered that the internal ear was filled with a liquid. Still later, it was found that peculiar, delicate structures are contained within this liquid.

Theories of "how we hear" have been built up on the basis of these structures and the fluid which surrounds them. Gross anatomy, comparative anatomy, and histologic anatomy have contributed to these theories. In addition to the anatomic structures themselves, certain physiologic facts became demonstrated. From the standpoint of the ear as an organ of hearing, the study of its function appears to be largely a problem of physics. Certain of these facts which have been revealed by studies in physics, have been considered in the previous communication. In addition to these facts that have been brought forth by studies in anatomy and physiology, some evidence has resulted from a study of lesions of the cochlea. Such pathologic evidence includes studies of cases of tinnitus and diplacusis, observations in cases of otosclerosis, and animal experimentation.

The answer to "how we hear" must be consistent with the anatomic structure of the internal ear, with the observed

*See December (1925) number of THE ANNALS.

physiologic facts, and with the pathologic evidence. Up to the present time no entirely satisfactory theory has been advanced. However, certain theories are consistent with the anatomic structures and also account for many of the observed facts.

It would seem established that in the normal ear the sound waves enter through the oval window and continue as sound waves through the endolymph of the cochlea. Here the question arises as to how these sound waves are transmuted into nervous impulses. Is it by direct stimulation of the nerve cells or is there an intermediary vibrating structure which produces a stimulation of the terminal filaments of the auditory nerve? If there is a vibrating mechanism, does this mechanism vibrate in its various parts or does it vibrate as a whole? If this vibrating structure vibrates in its various parts, do these various parts respond to wave motions of various frequencies? Does a single vibrating structure vibrate to a wave motion of certain definite frequency or do several contiguous vibrators respond? Is the analysis of tone accomplished by the cochlea or by the cells of the cerebral cortex? Are separate cortical centers stimulated by separate impulses transmitted by separate nerve fibers, or is it the rapidity of arrival of the nervous impulses, coming over the same nerve fibers that determines the pitch sensation of any given tone?

The question as to "where we hear" may be answered as follows: The conscious sensing of hearing is undoubtedly a function of the cerebral cortex. Experimental work on dogs and monkeys and the study of a few deaf patients who have shown gross lesions at autopsy have suggested that there is a cortical area for hearing in the first and second temporal convolutions of each side. Further, it is plausible to consider that the cochlea changes the physical vibrations of sound waves into corresponding nervous impulses. The essential problem, therefore, is to determine how the cochlea accomplishes this change. The anatomic structure of the cochlea makes it highly probable that it acts as a vibrating structure; that each physical vibration from the external world causes a corresponding vibration within the cochlea; and that the cochlea then transmits to the brain a corresponding nervous impulse.

The above conceptions are generally regarded as plausible. Most investigators will agree up to this point. The difference in opinion arises as to the precise way in which the cochlea works. Nearly all consider that the cochlea possesses a vibrating mechanism. Histologic study demonstrates that there are two membranes, either of which might be regarded as the vibrating mechanism, the basilar membrane and the tectorial membrane. From a general broad standpoint, it is possible to consider that the essential vibrating structure consists of either of these two membranes. It is also possible that both of these membranes may cooperate as a vibrating mechanism. Each of these two membranes has certain characteristics which favor its being regarded as the essential vibrating structure. Detailed evidence favoring each of these membranes will be considered later.

Most authorities consider that the active mechanism vibrates selectively; that certain portions of the cochlea respond to certain vibrations, whereas other portions of the cochlea respond to other vibrations. Other investigators believe that the vibrating mechanism does not act selectively, but that each external stimulus causes the entire mechanism to vibrate as a whole.

The two theories of the response of the cochlea, just mentioned, suggest two physical analogies: The strings of the harp and the diaphragm of the telephone transmitter. In other words, it would seem to simplify this subject to make a general classification of all theories of audition under either the "harp" theory or the "telephone" theory.

THE HARP THEORY.

The "harp" theory is based upon the principle of physical resonance. This principle of physical resonance may be illustrated by a simple experiment: The harp may be used, or more conveniently, the piano. One sits at the piano and presses the loud pedal, thus releasing the dampers from all of the strings. If one then sings a tone which is accurately in tune with any string on the piano, that string will be set into sympathetic vibration. It will continue to vibrate after the voice has ceased. This vibration of the string gradually dies away to inaudibility. If several tones are sung into the piano, a

series of strings is set into vibration. Each string of the piano can thus be made to vibrate either by itself or in combination with any number of other strings. Each string, depending upon its length, tension and mass, is set into vibration by a wave motion of a particular frequency. In other words, the frequency which excites this string is the same frequency which the string itself would emit if it were plucked or struck. If one sings the tones C E G C^1, the harp or piano will faithfully reproduce these same tones, C E G C^1. Each string, as it were, stands ready to respond to its own appropriate frequency. This is the principle of physical resonance. Although the harp or the piano is made up of strings which produce only tones and semitones, they are capable of reproducing a very complex sound. For example, if one shouts into the piano it will "shout back" with remarkable mimicry.

The "harp" theory postulates that the cochlea is like the harp of the piano: Vibrations in the external world, of a definite frequency, produce a sympathetic vibration of certain strings or vibrating elements within the cochlea. In the same way, any combination of vibrations in the external world produces a corresponding combination of vibrations of the appropriate vibrators within the cochlea. In this way, for every external wave impulse, whether simple or complex, there is a corresponding pattern of sympathetic vibrations within the cochlea. This "harp" theory was first given practical expression by Helmholtz, although it appears that the theory had been advanced previously, notably by Perrault in 1680, Duverne in 1683, and Cortugno in 1760. It is usually spoken of as the "resonance theory."

Helmholtz considered at first that the vibrating elements were in the organ of Corti. He also considered that each vibrating element is associated with a separate nerve filament. Several other investigators, although accepting the resonance, or "harp," theory, have differed from Helmholtz as to which structures of the cochlea constitute the vibrating elements. Measurements of the basilar membrane led Hensen and others, including Helmholtz, to regard the basilar membrane, and not Corti's organ, as the vibrating structure. Their studies indicate that the basilar membrane, throughout the entire length of the cochlea is made up of transverse fibers. The longest

and most loosely stretched fibers are at the apex of the cochlea. The shortest and most tightly stretched fibers are at the base of the cochlea. Hence the portion of the basilar membrane near the apex is adapted to vibrate in response to vibrations of low frequencies. The portion of the basilar membrane near the base of the cochlea is adapted to respond to vibrations of high frequencies. Both Hartridge and Wilkinson have presented plausible evidence that the lengths and the tensions and the masses of these transverse fibers are consistent with this conception of the function of the basilar membrane.

On the other hand, Shambaugh, Hardesty and Kishi proposed that the tectorial membrane, and not the basilar membrane, is the vibrating structure. Shambaugh has given definite evidence against the basilar membrane as the vibrating structure. He has shown, histologically, that as it is the basilar membrane that contains the nutrient artery, this varying vessel and its blood contents render it less fit than the tectorial membrane to act as a vibrator. Further, Shambaugh has found, histologically, a well formed organ of Corti, but a complete absence of the basilar membrane in the lower coil of the cochlea. In one of his specimens the Corti's cells rested upon a bony structure, which certainly could not vibrate. In all instances where he found an organ of Corti, he also found a tectorial membrane. He concludes, therefore, that the stimulation of the hair cells is accomplished without the intervention of a vibrating basilar membrane, and that the stimulation of these hair cells is brought about by the vibration of the overhanging tectorial membrane.

Whether it is the basilar or the tectorial membrane which assumes the rôle of active vibrator, it would seem established that the auditory nerve impulse is produced by the action of the tectorial membrane upon the hairs which project normally toward the tectorial membrane. In other words, if the basilar membrane is the vibrating structure, these hair cells are pushed against the tectorial membrane. If the tectorial membrane is the vibrating structure, its vibration directly affects the hairs.

Hartridge considers it unnecessary to single out one of these structures as the sole vibrating element. He regards the basilar membrane as the essential vibrating structure, but considers that it may be influenced by the organ of Corti and

also by the tectorial membrane. He considers that the organ of Corti, the basilar membrane and the tectorial membrane are all coupled together, and that necessarily they must all be influenced by the vibrations within the endolymph.

The "harp" theory, in its primitive form, postulated the existence of discrete strings or fibers. Later, anatomists demonstrated the continuity of these cochlear structures. It was found that these structures do not exist as separate fibers. There are not only connection between the fibers, but both fibrous membranes, basilar and tectorial, continue as spiral structures, from the base of the cochlea to the apex.

On the basis of the primitive "harp" theory, it was difficult to account for the coexistence of great damping and great selectivity. This led to certain modifications and developments of the original "harp" theory. The anatomic demonstration of the continuity of the vibrating mechanism gave a more feasible explanation of this coexistence of high damping and great selectivity. The lateral connection between the fibers, as well as the interlocking of the basilar membrane, the Corti's organ and the tectorial membrane can account for the high degree of damping that is present in the cochlea. Great damping is ordinarily opposed to high selectivity. However, high selectivity can be accounted for by assuming that only the crest of the vibrating area is effective in determining the pitch sensation. Very probably, the most nearly adjacent fibers would also be set into a certain degree of vibration. The pitch sensations produced by the adjacent fibers perhaps are not sensed, because they are masked by the predominant sensation of greatest intensity. This conception led naturally to the theory of "maximal amplitudes." In this theory the perception of pitch is determined by the position of the maximal vibrations of the continuous membrane. Low frequencies would cause a maximal vibration of one portion of the cochlea, high frequencies would cause a maximal vibration of another part of the cochlea. This is the theory of Gray. Rolfe has shown that it is consistent with dynamic requirements. Fletcher has extended this theory and has shown that it is entirely consistent with many of the observed facts of audition. This theory not only enjoys the merits of other "harp" theories which also account for the lower and upper limits of pitch per-

ception, the different gradations of loudness and the different gradations of pitch, and the production of subjective tones, but it is particularly satisfactory in explaining the facts of auditory masking of tones. In other words, this theory of maximal amplitudes possesses all of the advantages of the original "harp" theory. but it also accounts for other phenomena which are not accounted for by the simple "harp" theory.

THE TELEPHONE THEORY.

The "telephone" theory, in its simplest form, postulates that the entire vibrating membrane of the cochlea responds to every type of vibration, from the lowest to the highest frequency. The cochlea simply transforms these physical vibrations into nervous impulses. The nerve impulses are then conducted "en masse" to the brain. It is the frequency of arrival of these impulses at the cerebral cortex, which determines the pitch sensation. For example, if fifty periodic impulses per second reach the cortex, there results the sensation of a tone of low pitch. If ten thousand, a tone of high pitch.

A single diaphragm of a telephone transmitter or receiver can reproduce with marvelous accuracy the most complex and rapid movements of an entire symphony orchestra. The "telephone" theory owes its origin to the concept that the cochlea can act in the same way as the vibrating diaphragm of the telephone transmitter or receiver.

Another physical analogy is found in multiplex telephony, and also in radio broadcasting. It has been found that it is possible to send many telegraphic and also telephonic messages over the same wire, simply by having the different messages transmitted by different "carrier freqeuncies." Thus, this one wire is capable of transmitting many messages at the same time. Also in radio transmission many stations can be on the air at the same time. The "telephone" theory, in this way, postulates that many varied nervous impulses are transmitted over the same nerve tracts, from the entire cochlea to the cerebral cortex.

It is obvious that this theory is quite different from the "harp" theory. Instead of regarding the cochlea as an organ whose separate parts respond to different frequencies, the "telephone" theory simply regards the entire cochlear mem-

brane as vibrating as a whole. In other words, when vibrations occur in the endolymph, regardless of their frequency, the membrane is considered as being affected throughout its entire length. It simply acts as a relay. According to this theory, the cochlea merely changes physical wave motions into nervous impulses of a corresponding frequency. The same auditory termini and the same auditory filaments transmit all the varied vibrations to the brain. According to the "telephone" theory, therefore, there is not a separate vibrating element nor a separate nerve filament for each tone that can be sensed. The chief exponents of the "telephone" theory include Rinne, Voltalini, Rutherford, Waller, Ewald, ter Kuile, Meyer and Watt.

There are various modifications of the "telephone" theory which have been suggested. One is that vibrations of small amplitude penetrate only a short distance into the cochlea and cause vibrations of only that portion nearest the oval window. As the amplitude of the vibrations becomes greater and greater, there is a deeper penetration into the cochlea and finally the vibrations of greatest amplitude cause the entire cochlea to vibrate. Others have suggested that vibrations of high frequency actuate only a small portion of the cochlea, whereas vibrations of low frequency actuate the entire cochlea. Another ingenious theory is that of Wrightson, who expands the original "telephone" theory. He considers that each pendular vibration reaching the ear is capable of producing four distinct types of nerve stimulation. As a technical engineer, he utilized this idea to account for the presence of subjective tones. Previous to Wrightson's explanation, on the basis of the "telephone" theory it had been difficult to account for the unquestioned occurrence of these subjective tones.

RELATIVE PLAUSIBILITY OF THE TWO THEORIES.

In an attempt to weigh the evidence there are certain facts and arguments which appear to lend credence to each of these two theories. First, the "telephone" theory. The essential merchanism consists of a cochlear tube with the oval window at one end and the round window at the other end. The sound waves which reach this cochlear tube originate in the external world as alternate condensations and rarefactions. Each series

of these vibrations causes a succession of condensations and rarefactions to move the tympanic membrane and thence the stapes. When the effect of a condensation reaches the stapes it is pushed inward. When the effect of a rarefaction reaches the stapes it is drawn outward. According to this conception, when the stapes is pushed inward the entire cochlear fluid is displaced toward the round window, and the round window bulges outward. When the stapes is drawn outward the entire cochlear fluid is displaced toward the stapes and the round window is drawn inward. This conception favors the "telephone" theory because it would seem probable that a gross movement of the entire cochlear fluid must necessarily affect the entire membranous structure of the cochlea. A gross vibratory motion of the entire cochlear fluid would most probably cause a synchronous displacement of all of the contained membranous structures.

Again, it has been mentioned as an anatomic fact that no membranous fibers exist separately within the cochlea. The very continuity of these fibers makes it difficult to consider that any portion of the cochlea could vibrate without setting into vibration at least the adjacent parts of the cochlear structure.

Again, it is easier to account for the damping of tones on the basis of the "telephone" theory. It is well known that an individual ceases to hear a sound almost immediately after the cessation of the flow of sound energy into the cochlea. The external vibrations cease, and apparently the sound sensation ceases at once. If a tone is introduced into the ear by a tube connected to the stem of a tuning fork, the sound ceases the instant the tube is closed. There appears to be little or no reverberation within the cochlea. There is no persistence of the tone, after the source is stopped. This manifest damping is obviously of a high degree, because otherwise sounds, such as articulated speech and music, would all "run together." Now, the vibration of the entire membrane in the fluid of the cochlea would experience much greater damping than would the vibration of separate fibers. For example, a plucked string continues vibrating much longer than a struck bass drum. This difference would be even more emphasized if the string and the bass drum were vibrating in a liquid medium. The

great degree of damping that does exist in the ear can, therefore, be more easily accounted for by considering that the cochlear structures do vibrate as a whole. On the basis of the "harp" theory, it is difficult to account for this very great damping which occurs on the perception of sound. Again, on the basis of the "harp" theory, it is necessary that the vibrating structure be "highly tuned," that is, only one fiber of a very restricted group of fibers must respond to vibrations of a certain frequency. Otherwise, vibrations of discrete frequency would be heard not as a pure tone, as it apparently is, but as a hazy mixture including contiguous tones. It would seem, therefore, as already mentioned, that the vibrating structure must possess two physical characteristics: a high degree of damping and a high degree of pitch selectivity. There are no analogies of physical vibrators which possess both great damping and high selectivity. These two properties are not compatible. Thus, the tuning fork when free to vibrate in the air, will be set into sympathetic vibration when, and only when, it is stimulated by a wave motion which has precisely the same frequency as the fork itself would emit. It will, however, respond vigorously to its own characteristic frequency. Now, if this same fork be placed in a viscous medium, such as oil or molasses, its vibrations naturally will be greatly damped, but in addition it can also be set into vibration by other tuning forks that are not of its own frequency, but are both above and below its own frequency. In other words, the tuning fork in the medium of the air responds only to vibrations of its own precise frequency; the tuning fork in the oil or molasses responds to vibrations not only of its own frequency, but to vibrations both below and above its own frequency. Yet the fork in the air, which is very selective in its response, is damped only slightly by the air. The tuning fork in the molasses is not selective in its response, but is greatly damped. Thus, the two properties of great damping and great selectivity are actually not found together in physical vibrators. Now these are the two properties which, in the "harp" theory, must coexist in the same vibrating mechanism of the cochlea. The "harp" theory, therefore, has difficulty in reconciling this actual phenomenon of the coexistence of great damping and high selectivity.

One of the arguments against the "harp" theory is that diplacusis is found so infrequently. We know that individual strings of a piano get out of tune very easily. Slight changes in temperature and humidity are sufficient to put them out of tune. The strings of a violin, especially those made of catgut—an organic material—do not stay in tune more than half an hour. It is imposing a difficult requirement upon the vibrating structures of the cochlea to assume that the individual vibrators in one cochlea should keep in tune with the corresponding vibrators of the other cochlea. In other words, it is difficult to see why there is not more diplacusis than there actually is, if each part responds to a particular frequency. Given any external sound vibration, in almost all individuals, the two cochleas respond to it exactly the same way with regard to pitch. According to the "harp" theory, each frequency affects a particular vibrator in one cochlea and a corresponding vibrator in the other cochlea. These two vibrators, one in each cochlea, must agree precisely in their length, tension and mass. The slightest change in any of these properties would presumably produce a diplacusis, according to the "harp" theory. It is difficult to conceive that all of the corresponding structures in each cochlea should maintain these exact physical characteristics throughout the lifetime of the vast majority of individuals.

Finally, in considering the evidence that favors the "telephone" theory, either directly or by presenting difficulties for the acceptance of the "harp" theory, anyone who gives thought to this subject is impressed with the unusual requirements of length, mass and tension, which the vibrating structures must possess if they respond as individual vibrators. Those who favor the "telephone" theory maintain that the fibers of the cochlear structures are inadequate to respond to the extreme low and to the extreme high frequencies. They point out that it is particularly difficult to explain how a fiber only .5 mm. in length can respond to a vibration of 20 d. v. Throughout the cochlea the fibers do not differ greatly in length. The shortest fibers are approximately .16 mm. and the longest fibers are only about three times this length. In other words, we have to account for a range of pitch extending over ten octaves and yet the variation in length is sufficient to account

for only slightly more than one octave. Of course the tension and the mass may vary; but the mass is only approximately known and the tension cannot be accurately determined.

As an illustration of the above, if the mass and the tension of all fibers are the same, the fiber which responds to 20 d. v. would have to be 1,000 times as long as the fiber which responds to 20,000 d. v. That is to say, the fiber within the cochlea that is concerned with the lowest tone would have to be 1,000 times as long as the other fiber within the cochlea that is concerned with the highest tone. Since the variation in length is not one thousandfold, but is only threefold, a heavy burden is placed upon the properties of tension and mass in order to render the cochlear fibers adequate for such an extensive function.

If the length and mass are the same for all fibers, the tension of the 20,000 d. v. fiber must be 1,000,000 times as great as the tension of the 20 d. v. fiber. Such an extreme variation of tension seems improbable.

Again, if the tension and length of all fibers are the same, the mass of the 20 d. v. fiber would have to be 1,000,000 times the mass of the fiber for 20,000 d. v.

Of course, the length, the mass and the tension may all vary in the actual structures of the cochlea. However, even granting the most plausible extent of variation in these three factors, it is not easy to reconcile them on the basis of the "harp" theory. If the longest fibers are only three times as long as the shortest ones, the tension and mass of the different fibers would each have to vary at least three hundredfold. Such a wide range of variation of mass and tension is more than most authorities will admit.

The arguments in favor of the "telephone" theory and against the "harp" theory, therefore, are: The mechanism of the stapes and the cochlear tube is adapted for gross displacement of the entire membranous cochlea; the continuity of the cochlear membranes, from the base to the apex, suggests that the vibrating membrane acts as a diaphragm rather than as individual fibers; the assumption that this continuous membrane vibrates as a whole, makes it easier to account for the very great damping of tones that does occur within the cochlea. In addition, the "harp" theory confronts the diffi-

culties of the infrequent occurrence of diplacusis and also the extraordinary requirements of the length, tension and mass of the receptors within the cochlea.

Arguments in favor of the "harp" theory include the following: With regard to what has just been stated in reference to the length, tension and mass of the vibrating structures of the cochlea, both Hartridge and Wilkinson, assuming that the basilar membrane is the active vibrating mechanism, have presented the results of careful calculations and physical experiments which present plausible evidence that the actual fibers of the basilar membrane can be regarded as possessing the necessary requisites of length, tension and mass to satisfy the "harp" theory. The evidence of Hartridge relates especially to the factor of the tension. He cites instances of tension in various animal tissues, comparable to the tension that would be demanded by the "harp" theory. Assuming generally accepted values for the length and the mass, he calculated what the tension must be in the cochlear fibers in order to respond to vibrations of the lowest and the highest audible frequencies. He then demonstrated that this variation of tension was possible in similar animal tissue. In other words, realizing that the length of these fibers is fairly well determined, and that the mass of the fibers is approximately known, he realized that the burden of the proof rested upon the matter of the tension. The question was whether these cochlear structures would hold up under the tension required to meet the needs of the "harp" theory. His experiments indicate that the tension of the cochlear structures can be regarded as adequate. The histologic work of Gray throws additional light on the matter of tension. He notes that the ligament which constitutes the outer attachment of the basilar membrane is much more fibrous and bulky at the base than at the apex. This suggests that nature has provided for a relatively great tension at the base and a gradually diminishing tension as the apex is approached and the least tension at the apex. This ligament to which the basilar membrane is attached is five times as thick at the base as it is at the apex.

Wilkinson reasons that the different vibrating structures within the cochlea are differently "loaded." This "loading" is due to the cochlear fluid itself. He argues that those high

frequencies which would affect the structures in the base of the cochlea, would have a ready exit at the adjacent round window, whereby only a small portion of the cochlear fluid would be involved in the actual vibration. Low frequency vibrations, on the other hand, affecting the structures in the apex, would have to set into oscillation the full length of the cochlear fluid between the apex and the round window. He regards the cochlear fluid as constituting the main element in the factor mass. As different portions of the active membrane are set into sympathetic vibration, they set into similar vibration the adjacent portions of the cochlear fluid. At the base this mass factor is least; at the apex the mass factor is greatest. For the appreciation of a low frequency the requirements are great length, slight tension and great loading, or mass. For the appreciation of high frequency the requirements are short length, great tension and slight mass. The works of Hartridge and of Wilkinson have, therefore, made it plausible to consider that the fibers of the basilar membrane can meet these extraordinary requirements.

Shambaugh, and also Hardesty, have presented evidence which indicates that the tectorial membrane is constructed in such a manner that it could respond selectively to audible frequencies. Shambaugh has shown that the tectorial membrane possesses a lamellar structure, and it varies with respect to both length and mass from the base to the apex. He has shown that the tectorial membrane is smallest at the base, and gradually increases in size until it attains its greatest dimensions at the apex. Hardesty has shown that there is a 4.8 fold width variation in the tectorial membrane of the pig. According to his measurements, it varies from 45.6 microns at the basal end to 220 microns near the apex. He has shown that the thickness, which determines the mass, is greatest at the apical end, and diminishes toward the base. His measurements give a thickness of 74.7 microns at the apex and 13.4 microns at the base. The above evidence suggests that either the basilar or tectorial membrane can satisfy the requirements of the "harp" theory.

It is a noteworthy fact of audition that the human being is able to analyze complex tones into their simple tone com-

ponents. This must be accomplished either centrally or peripherally—either in the cerebral cortex or in the cochlea. The complex structure of the cochlea favors the concept that it, like the eye, possesses an analytic function. If the analysis of a complex tone into simple tone components is accomplished by the cochlea, as brought out by Helmholtz, the principle of sympathetic vibration is the only known means by which this remarkable function can be explained. The simplest example of a complex tone is furnished by any musical instrument, such as the violin or oboe. One of the demonstrated facts of audition is that the quality of a tone is determined by the fundamental frequency, and the number and prominence of the harmonics or overtones which accompany the fundamental tone. For example, when one listens to the tone of a violin, he appreciates not only the fundamental, but also a large number of the overtones. Some highly trained ears can actually recognize the presence of many of the individual overtones. Even an untrained person, with the aid of appropriate resonators, can recognize the presence of many of these overtones. The assumption that different parts of the cochlea respond to vibrations of different frequencies would, therefore, seem sufficient to account for the appreciation of these individual tone components, without throwing the burden of analysis on the cerebral cortex.

Hartridge records an experiment which lends favor to the resonance hypothesis. By an ingenious arrangement of the siren he is able suddenly to produce a reversal of phase in the sound vibrations issuing from the holes in the siren. Such a reversal of phase produces a momentary cessation, and then a building up of the same tone sensation to its initial loudness. He found that this could be done for all the tones he could produce with the siren. Those "telephone" theories which assume that the action of the cochlea is "piston like"—that is, which assume that the fluid and membrane within the cochlea follow the motion of the stapes in a "deadbeat" fashion —would encounter quite impossible difficulties in an attempt to explain this phenomenon. The frequency of arrival at the cerebral cortex of the successive impulses would not be interrupted sufficiently to cause a cessation of the tones. Even those "telephone" theories which assume that there is an active

vibrating membrane within the cochlea, which is not strictly "deadbeat," would encounter difficulties in explaining this observed fact. On the other hand, it can be explained satisfactorily upon the assumption that there is a series of individual vibrators within the cochlea.

There is a generally accepted theory that has direct bearing upon this problem of the nervous mechanism of the cochlea, called by some the "Neuron Theory." The fundamental Neuron Theory postulates that the nervous system is made up of discrete units, called neurons. There are further elaborations of this simple concept, which also are rather generally accepted.

First from the qualitative standpoint, it is considered that each neuron produces its own characteristic response, irrespective of how it is stimulated. The stimulus may be mechanical, thermal, or electrical, and may be applied through natural or artificial means, and yet there results only one characteristic type of response. For example, as applied to vision, if the optic nerve is stimulated by any form of irritation there results a visual sensation and nothing but a visual sensation.

From the quantitative standpoint, it is now considered by most authorities that neural activity follows an "all or none" law. If the neuron acts at all it acts with its full intensity. Each neuron gives rise to a discrete amount, or quantum, of response. According to this concept the intensity of any response is determined solely by the number of neurons involved.

If we are to consider that the above theories of neural action are accepted, it would seem that any theory of the function of the cochlea must come under these general theories and be subject to their postulates. This would suggest that it would be impossible for the receptors within the cochlea to act in any way except selectively, and that each receptor would send its own characteristic qualitative and quantitative stimulus to the cerebral cortex. In other words, applying these general concepts to the problem of "how we hear," when certain neurons of the auditory nerve are stimulated, there results a sensation of low pitch; when certain other neurons of the auditory nerve are stimulated there results a sensation of high pitch. The intensity or loudness of the sensation is determined

by the number of contiguous neurons which are stimulated. On this basis, a feeble tone sensation results when only a few neurons are excited; a correspondingly more intense sensation results when a larger number of contiguous neurons are excited.

With the above in mind it is of particular interest, therefore, to see how these general concepts of neural activity actually fit in with the facts of histologic anatomy and the precise determinations of auditory function. According to the best authorities on the histology of the cochlea and the VIII Nerve, there are approximately 15,000 separate hair cells in the organ of Corti and also 15,000 nerve filaments in the auditory branch of the VIII Nerve. Not only do we have the 15,000 cells and filaments, but we have also the mechanism for exciting a few, or many, of these hair cells. A vibration of a certain frequency and intensity would produce a vibration of a circumscribed area of the active membrane. The location would be determined by the frequency of the vibration. The extent, by the amplitude. Whether it be the basilar membrane or the tectorial membrane, or both, which participates in the vibration, the number of neurons which are stimulated would be proportional to the area which is set into vibration. Hence, as the vibration increases in intensity a larger and larger area would be set into vibration, thus exciting a correspondingly larger number of neurons.

In this way, it is possible to account for the analytic function of the cochlea in appreciating all gradations of pitch and loudness. According to the experimental work of one of the writers the cochlea can sense about 1,500 gradations of pitch and about 240 gradations of loudness. On the basis of 15,000 individual receptors and the 15,000 individual nerve filaments, there would be ten receptors and filaments for each pitch which the normal cochlea can sense. When a loud tone of given pitch is sensed it would seem probable that not only the ten allotted receptors and filaments are excited, but that a large number of contiguous vibrators and filaments are also excited. In this connection it is not without interest to note the following simple observation: When one listens to a loud tone which is pure, physically, one is conscious that the tone is somewhat "blurred" or "hazy." That is, the tone seems to include cer-

tain adjacent frequencies, rather than vibrations of a single frequency.

The "harp" theory, that is, a selective action of the cochlea, would therefore seem to be supported in that Nature has provided a sufficient number of nerve receptors and filaments to account for the appreciation of all the possible gradations of pitch and loudness.

Certain pathologic evidence supports the "harp" theory. This evidence includes tonal tinnitus; diplacusis; tonal depressions; invasion of the cochlea from otosclerosis, and animal experimentation.

Tinnitus frequently is characterized by a definite tonal property—the subjective tone is of a precisely determinable pitch. One of the writers produced a tonal tinnitus in his own ear by listening to an excessively loud tone for a period of ten minutes. This tinnitus persisted continuously for six days. A tonal tinnitus has been observed by the writers, and its pitch and loudness measured, in an individual who otherwise has normal hearing. Precise measurements revealed perfect cochlear function in each ear, with the single exception of this tinnitus which was of a pitch of 4,500 d. v., and a loudness of 18 sensation units. Another patient showed almost normal hearing for all tones up to 1,600 d. v. There was a precipitous drop in the acuity at this point; from then on upward there was almost complete loss of function. The patient exhibited a tinnitus which was of the same frequency at which his deafness began—1,600 d. v. The pitch of his tinnitus was at the pitch threshold where this precipitous change occurred from good hearing into poor hearing. Such phenomena of tonal tinnitus are more satisfactorily accounted for if we assume that the cochlea responds with pitch selectivity in its different parts. In brief, it is a fact of audition that many internal ear lesions produce tinnitus. This tinnitus is often limited to a narrow range of pitch or even to a pure tone. It would appear probable that such a lesion in the cochlea affects certain vibrators or a certain circumscribed group of vibrators in the cochlea.

Similarly the phenomenon of diplacusis, in which the two ears hear the same physical vibration as tones of different pitch, is more easily explained on the basis of a lesion of

certain definite parts of the vibrating structure. In certain patients the diplacusis, or double hearing, is noted throughout the entire range; however, in other patients the double hearing is limited to only certain frequencies. It is difficult to account for this phenomenon on the basis of the "telephone" theory. It is much more satisfactory to consider a peripheral rather than a central explanation.

Recent precise measurements have seemed to indicate that "tonal islands" are not so frequent as previously supposed. These socalled "tonal islands" appear to be, more strictly, tonal depressions. The writers have not found one instance of complete deafness over a narrow pitch range, with hearing present above and below this pitch range. Tonal depressions, however, do occur. Apparently they are explicable only by some form of "harp" theory. Thus, the existence of these tonal depressions, which in some instances are marked, suggests a disturbance of a certain particular portion of the cochlea which, presumably, presides over this particular pitch range.

The nature of the invasion of the internal ear in otosclerosis suggests that the cochlea acts selectively. The following has frequently been observed, particularly since instruments of precision have been used for charting the hearing: (1) The low tones are heard less and less well, whereas, the high tones are heard very well; (2) As the disease progresses, the low tones are heard less well, the tones in the middle register are depressed relatively little, whereas, the high pitched tones are depressed markedly. In certain far advanced cases of otosclerosis there is total loss of hearing for all tones above 2,000 d. v. These observations suggest that the process of spongification of the bone around the oval window has extended into the adjacent first turn of the cochlea where, it has been assumed, the high pitched tones are sensed.

Experimental work on animals by Siebenmann, Yoshii, Wittmaack and Marx has shown that if a guinea pig be subjected to sound vibrations of great intensity for a considerable period, subsequent histologic examination shows degeneration in circumscribed areas within the cochlea. Certain of these experiments have indicated, not only that certain parts of the cochlea are affected by such stimulation, but specifically, that subjecting

animals to tones of high pitch, produced alterations in the structures at the base of the cochlea. It has also been shown that tones of medium pitch produced alterations in the middle turn of the cochlea and that low tones produced alterations in the apex.

In conclusion, at the present time the weight of evidence seems to be in favor of some type of "harp" theory. The particular theory that appears most reasonable is the theory of maximal amplitudes, which postulates that there is a series of resonators within the cochlea very closely coupled so that one cannot act without causing its immediate adjacent structures to participate in the vibration. From the standpoint of the clinical otologist, this theory is the most satisfactory. It is consistent with the known anatomy, gross and histologic; it is consistent with the continuity of the vibrating structures within the cochlea; it is consistent with the observed facts of audition. In addition, it offers to the clinician the most satisfactory explanation for the familiar pathologic conditions which he meets in routine practice.

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IX.

CONGENITAL OCCLUSION OF THE CHOANÆ.*

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"The choanæ are communicating passageways—not mere orifices—between the nasal fossæ and the nasopharynx." They are very narrow at birth and infundibuliform in shape. The bony walls cause the normal nasopharyngeal communications to be permanently open and free, unless blocked by some outside structures, such as adenoids.

The choanæ may be closed congenitally. The occlusion may be membranous or bony, unilateral or bilateral, complete or incomplete. Richardson[1] classifies congenital occlusion of the choanæ as follows:

1. Membranous.—This is formed posterior to the nasal cavities in the nasopharyngeal cavity, but in contact with the postnasal orifices so as to completely obstruct them. The membrane extends from the upper surface of the soft palate, where the hard palate joins the postnasal cavity.

2. Osseous.—The bony plates are usually vertically placed —concave pharyngeal aspect—within the nasal chamber a millimeter or more from the free border of the postnasal orifices.

3. Developmental.—The bones entering into the formation of the postnasal orifices become united, thus more or less completely obstructing the postnasal orifices.

Congenital complete bilateral occlusion is very rarely observed, for the reason that most babies with this affliction die very shortly after birth. Mouret[2] affirms that he has been able to find but four authentic records of such babies who lived. I have one additional case to report today. Many such cases were reported as complete occlusion, it is true, but in his opinion they were really incomplete ones.

Up to 1923 Libersone[3] found reported 170 cases of all types. Probably McNaught[4] is correct when he maintains that this

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condition is very much more frequent than has been generally supposed. One reason for believing this is that cases of unilateral occlusion are occasionally discovered in adults who did not suspect that they had any such trouble or who were so little annoyed by it that they did not seek relief. Cases of bilateral occlusion have been discovered at 14 and 16 years of age. Cases of asphyxia neonatorum are at times caused by congenital occlusion of the choanæ; occasionally this is recognized, but more often it goes undetected.

There are certain coexisting factors in connection with this condition that are very well worthy of mention. It is interesting to note that females are afflicted in this way fully twice as frequently as males, according to the best statistics that are obtainable upon the subject.

So far as we have been able to determine, it is not hereditary, although there is on record one family, six members of which were affected. It consisted of a mother, her two sisters, her two daughters and a son.

Quite frequently it is associated with other deformities, although in most cases they are deformities which it may perhaps produce, such as high arched palate. At other times it is associated with other congenital defects—coloboma of the iris, for instance.

When it is unilateral, it more frequently occurs on the right side; but it occurs three times as often bilaterally as it does unilaterally. Ninety per cent of the cases are said to be of the complete, bony type.

The pathogenesis of this condition is very interesting, and there are various views upon the subject, some of which are conflicting. Jacques[5] reports a case which he believes was due to congenital lues. Trauma at birth was mentioned by Ullman,[6] but he abandoned this view because it could be caused only by a fracture—and a fracture of the choanæ at birth is practically impossible. Others believe that this is caused by a hyperplasia of the bony skeleton of the posterior nares, because of the finding of a small opening in the upper section of the choanæ near the septum. Some investigators believe that the vertical plate of the palate bone is the one that proliferates (Bitat, Kundrat, Bennett,[7] Schroetter, Hopman,[8] Boty, Ricardo and Chiari); whereas a different school main-

tains that the horizontal plate causes the trouble (Luska and others). Still a third school is of the opinion that it comes from an overgrowth of the vomer.

Among those who are of the opinion that it is caused by a persistent bucconasal membrane may be numbered Shaeffer,[9] Bonly, Hoag, Hochheim[10] and Dusque. Decuring thinks it is a deformity of the soft palate.

A review of the embryology, according to Schaeffer, may be enlightening. "The characteristic epithelium of the nasal areas is recognizable as early as the third week of embryonal life. During the fourth week these areas become passively depressed by a positive increase in the thickness of the surrounding mesoderm which pushes the overlying ectoderm into relief. The nasal pits are thus formed. In a thirty-five days embryo the nasal pits have deepened sufficiently to partake of the nature of cleftlike fossæ which end blindly at their dorsal and inferior termination. This is the primitive nasal fossa, and its dorsal growth meets the ectoderm of the oral fossa and forms the bucconasal membrane. This membrane is so attenuated in a thirty-five to thirty-eight millimeter embryo that rupture usually ensues and the primitive choanæ are formed which establishes the communication between the nasal fossa and the oral cavity and also delineates the primitive palate. Lack of this rupture leads to atresia of the choanæ as a result of an organization of the epithelial plugs which occasionally block the choanæ, rarely osseous tissue develops in the organization.

These primitive choanæ do not correspond in position to the definitive choanæ, which are placed farther dorsal and are established in the third month when the definitive palate is completed."

Ecker and His[11] do not concur with Schaefer. It is their belief that as soon as the nose and mouth are formed in the human fetus, there is a connection between them; that this connection does not put in an appearance at a later period of development. But they agree with Schaefer that the nasal cavities later are partially shut off from the buccopharynx by the growth inwards of the hard and soft palate.

Hochstetter,[12] Tiemann[13] and Keibel[14] are of the same opinion as Schaefer, holding that in cases of choanal atresia

it is possible for the nasobuccal membrane to become permeated by mesoderm, in which event it fails to break down.

Fraser[15] directs our attention to the fact that the theory of Schaefer and those who concur with him is in line with our knowledge of the development of the vagina, anus and the external auditory meatus. He also affirms that in the majority of cases of choanal atresia the occluding membrane is well in front of the choanæ, which lends support to the theory that congenital atresia is due to the failure of the nasobuccal membrane to break down.

Frazer[16] gives support to the theory of Hopman to the effect that the choanæ may be narrow enough to produce a stenosis. But Hopman ascribes this narrowing to the presence of an asymmetric fetal anlage, and he reports a number of cases in support of his contention that need not be cited here.

Mouret[2] refers to Professor Vialleton, when he asserts that the two gutters appearing in the roof of the mouth, connecting the nose and the mouth, are improperly called the primitive choanæ. Physiologically, they may be so, he grants, but not topographically or anatomically, for the reason that the definitive choanæ are farther back and lie in the opposite direction. Professor Vialleton further maintains, according to Mouret, that the nasal cavities owe their growth to a proliferation of epithelium. If this proliferation does not progress in a normal manner, there occurs an occlusion of the choanæ. He does not consider the persistence of the bucconasal membrane an etiologic factor in such cases.

We have seen that the authorities cited above are not in complete accord. It is the belief of the present writer that the cases that fall in group two, given above, are very likely caused by a persistent bucconasal membrane, because the occluding plate, or membrane, is well in front of the choanæ. Those that are under group three are probably caused by narrowing of the choanal canal.

There are two other points that properly should be considered at this time. The first is that the persistence of the bucconasal membrane would not necessarily interfere with the fusion of the palatine processes. Accordingly, any deformity of the palate is independent of the condition of the choanæ. The second fact to which I desire to direct your attention is

that choanal occlusions, whether bilateral or unilateral, do not always, of necessity, produce a high arched palate.[19] In other words, high arched palates are not necessarily the result of mouth breathing.

The comparative anatomy of the choanæ and the evolution of nasal breathing is of some bearing on the subject (Negus).[21] In the majority of fish there is found to be no communication between the simple nose and the food tract. In dipnoi, however, such a communication does exist. In these fish inspiration is affected by the swallowing of air through the mouth, as occurs in some salamanders and newts. While the air breathing proceeded there would therefore be no circulation of air through the nose, and the olfactory sense would not be acute, therefore a communication or choanæ is present in the roof of the mouth, whereby air can be made to pass over the olfactory rosetti, so as to give off odor sufficient for perception of scent. In these cold blooded animals there is no necessity for air to pass through the nose as far as respiration is concerned.

Thus air enters in very low types by the mouth, nasal breathing being a secondary modification of respiration, and as the path of evolution is traced up there is to be found in all cases a continuation of this close relation of air tract and food passage. When nasal respiration is firmly established, there is still a necessity for the crossing of the two streams. In birds, Ingersoll[22] says the posterior nares have fingerlike projections which interlace and can shut off the nose from the pharynx and larynx.

The probable sequence of events in the evolution of nasal breathing is illustrated by the facts known of human embryology. The nasal area develops, the nasal pit appears, the bucconasal membrane breaks down and the palate is completed.

Congenital occlusion of the choanæ was first recognized by Otto in 1830 at postmortem. In 1853 Emmert[17] reported the first known case in a living patient, a child seven years of age, with a double occlusion. He operated and the result was a complete recovery.

Symptoms of bilateral complete occlusion are very striking and are so definite as to make the diagnosis an easy one and quite apparent. This is not true of a unilateral or a partial obstruction. When a complete occlusion is present, the infant

at birth has much difficulty in breathing. It is impossible for the baby to breathe through its nose, and it will not breathe through its mouth, because mouth breathing is an acquired habit. The tongue, being markedly developed for suckling purposes, increases the difficulty in mouth breathing. Accordingly, the mouth must be held open to relieve the difficulty.

Difficulty in nursing is always associated with the condition that we are discussing. It is necessary that the infant be fed with a spoon or by means of a stomach tube. As the infant grows older, it may partially overcome this symptom, but it is never able to take its entire feeding without difficulty, usually swallowing air with the food.

Another symptom is that the nasal cavities are always filled with a secretion of a peculiarly glary, gelatinous consistency, which irritates the lips and the nostrils. If the baby is held face down, this secretion will run out. It is unable to clear its own nose.

Anosmia is another symptom that always accompanies this condition. If it is discovered in early life and proper treatment is instituted, the sense of smell will develop normally. In older people the sense of smell has returned upon removing the choanal obstruction.

Some observers have noted that when the patient weeps, tears run out of the nose. Others have observed diminished lung expansion on the side of the nasal obstruction, an elevation of blood pressure, incontinence of urine, dyspepsia, nasal tone of the voice, dry pharyngitis and inability to indulge in violent exercise.

Very little information concerning this condition can be obtained by examination of the nose of an infant by anterior rhinoscopy; but I find it comparatively easy to see the posterior nares through the mouth by means of a Holmes nasopharyngoscope. Another method is the use of a palpating finger in the nasopharynx. It is impossible to pass a probe through the nose, which is very convincing proof of the posterior occlusion. Even when the procedures outlined above are followed, one cannot be absolutely certain of an occlusion without ruling out adenoids, hypertrophied turbinates, deflected septum, foreign bodies, specific adhesions, tumors of the nose or nasopharynx, polypi and acute infections.

There is only one well recognized method of treatment—making an opening through the obstruction and removing it, if possible. As the choanæ at birth are very small, particularly in their vertical diameter, no large opening can be made. The opening should be made as soon as possible after the diagnosis has been established, in order that the patient may be able to eat and may not lose his sense of smell. Even though a permanent opening is not made, the proceeding is still capable of giving a not inconsiderable degree of temporary relief. Richardson considers it hazardous to operate before the patient is at least two years of age, and Syme holds that any opening made before the expiration of the second year will not be permanent; but I find myself unable to agree with either of them. It is my opinion that one may safely operate at any age and that the opening, in most cases, will prove to be permanent.

This opening is more of a resection than a perforation, and it can be made by any cutting instrument of proper size, such as a trephine, drill, chisel, saw or biting forceps; but the secret of success lies in removing not only the occluding substance but the posterior portion of the nasal septum as well. Dan McKenzie first did this in 1910. This tends to prevent recurrences, as a large cavity is produced which remains open permanently. Many operators report that they have succeeded in maintaining the opening by means of rubber tubes placed through the nose and changed frequently for six months or more by running a strip of gauze through one nostril and out the other, retying and changing it daily (Brady[18]); but neither of these proceedings is necessary if the posterior portion of the nasal septum is removed.

At this point I will report two cases of congenital occlusion that have recently fallen under my observation, cases illustrative of the points that I have just been making. Dorothy A., aged four months, referred by Dr. Martin Nordland, was born two and a half weeks prematurely, labor being so long that it was necessary to make use of forceps. She was a blue baby, and breathing was established with considerable difficulty, the usual methods being of no avail. However, she began to breathe upon her mouth being held open forcibly. It was necessary to keep a nurse in constant attendance to hold the mouth open, as the minute her vigilance was relaxed the mouth closed,

breathing ceased and cyanosis appeared. Respiration with the mouth open was very noisy, and the tongue had a tendency to continuously cleave to the roof of the mouth. It was found necessary to feed the child through a stomach tube. After the eighth day, she learned of herself to hold her mouth open and to breathe in that way, but cyanosis occurred at intervals, being relieved by spontaneous opening of the mouth on the part of the patient. There was a profuse yellow discharge from both nostrils, which poured out when the infant was held face down. The voice was clear from birth, chest examination was negative and no thymus enlargements were disclosed by the X-ray. A very small mass of adenoids could be felt in the nasopharynx. By means of examination with the nasopharyngoscope, a choanal occlusion was plainly to be seen. It was dimpled on the right side and solid on the left. It was impossible to pass a probe through either nostril. Attempts to blow air through the nostrils with a Politzer bag caused tears to bubble at the lower punctum of the eyelids. There was no deformity of the face, jaw, nasal septum or palate.

The left posterior nares was opened by means of a chisel and the obstruction was found to be of solid bone, whereas the right posterior nares was obstructed by a substance that was part bone and part membrane. Both openings were thoroughly rasped with a frontal sinus rasp and the new openings were curetted. The posterior portion of the septum was removed as a whole by a forcep inserted through the mouth. Neither tubes nor packs were left in the nose. The patient reacted well to the operation, but no improvement in the breathing was discerned until after the second day. Then I ran a silver eustachian catheter through each nostril, well into the nasopharynx, at the same time holding the mouth closed. For the first time, the patient began to breathe through the nose, and she was able from then on to nurse normally, making use of the stomach tube unnecessary. During the next two weeks her breathing improved and she slept with her mouth closed. The yellow nasal discharge entirely disappeared. Six months later examination disclosed that she was breathing normally.

The second case was operated upon by my former associate, Dr. E. H. Parker. At that time the patient was seventeen years of age, and detailed a history of a right sided nasal

obstruction with profuse yellow discharge and loss of the sense of smell on that side. Two unsuccessful nasal operations had been previously performed. Her condition was diagnosed by Dr. Parker as unilateral congenital occlusion of the choanæ. He made a permanent opening through the occlusion. I saw her seventeen years later, when she was thirty-four years of age, and the opening was still functioning satisfactorily.

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X.

HYPERTHYROIDISM—ITS SIGNIFICANCE IN OTOLARYNGOLOGY.

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CLEVELAND CLINIC,

CLEVELAND.

Perhaps there is no disease regarding the etiology and management of which a greater diversity of opinion exists, or about which a larger literature has accumulated than is the case with hyperthyroidism. The condition is here denominated "hyperthyroidism," although as will be indicated below, this term is considered a misnomer by those who do not consider that the syndrome so designated is due to hyperactivity of the thyroid gland.

As for the etiology of the condition, I think that it can definitely be stated that it is unknown, most of the theories which have been proposed being concerned with the actively exciting factors of certain symptoms of the disease rather than with its primary cause. The great diversity of opinion regarding the primary cause of hyperthyroidism is indicated by the list of theories propounded by different authors as given in Bram's recent monograph—the bulbar theory, the intoxication theory, the kinetic theory, the thyroid insufficiency theory, the neurogenic theory, the toxic neurogenic theory, the dysthyroidism theory, the pluriglandular theory, the vagotonia or sympatheticotonia theory, and respectively, the neuroendocrin theory, the sympathetic, the thymus, adrenal, parathyroid, pituitary, gonad theories. There obviously is not time for me to enter into any discussion of the content of these theories, but in any case they have no bearing upon the purpose of this address, which has to do with the syndrome of hyperthyroidism once it has become established.

The general course of hyperthyroidism in contradistinction to the variety of causes which are attributed to it is strikingly

uniform. Gradually or rapidly increasing symptoms of general nervousness, tachycardia, tremor, disturbances of menstruation, and evidences of increasing vascularity of the thyroid gland are the outstanding signs. Barker gives the following concise picture: "We can scarcely do better than to urge the physician to think of the possibility of hyperthyroidism in every case in which he meets with (1) tachycardia, (2) rapid emaciation, (3) excessive sweating, (4) persistent watery diarrhea without apparent cause, (5) neurasthenic state, (6) outspoken lymphocytosis, (7) one or more of the eye signs, and (8) fine tremor." It will be noted that in neither of the above lists of symptoms of the disease is mention made of exophthalmos and that Barker mentions the eye signs among the last of the symptoms in his list. This seems to me to be an important note, for often a case of hyperthyroidism is missed because eye signs are not present, the term exophthalmic goiter having led too many of us to consider that that symptom is a sine qua non of the condition.

The acute type of hyperthyroidism needs no discussion. Its clinical course is outspoken and unmistakable. The subacute types are those which may require the finest clinical judgment for the establishment of the differential diagnosis. Among the conditions which might most readily be mistaken for hyperthyroidism are neurasthenia, neurocirculatory asthenia, early stages of pulmonary tuberculosis, paroxysmal tachycardia, cases of myxedema, even though that is definitely a condition of hyperthyroidism.

As for the general management of the disease, here again we find in general a uniformity of opinion that the one desideratum is the lessening of the thyroid secretion, but divergence of opinion as to the methods by which that end is to be attained, whether entirely by medical management, which includes the removal of whatever exciting or promoting causes of the syndrome are present—such as focal infections, sources of emotional or mental strain, excessive physical strain, and so forth, or actual diminution of the amount of the secreting gland by the X-ray or by a surgical operation. In the Cleveland Clinic the last cited is the method of choice for the reason that it is possible to control exactly the extent to which the activity of the gland is lessened, as is impossible when the

the X-rays are used. Other reasons for this choice which are at variance with the belief of many advocates of the X-ray cannot be given in detail here.

A word should be added regarding the incidence of hyperthyroidism and regarding certain characteristics of hyperthyroidism in children as they have an immediate bearing upon the special problem of this discussion. First, as to the incidence. We are so accustomed to consider that goiter and many diseases of the thyroid gland are in the main confined to certain fairly well limited goiter districts that we may overlook the fact that hyperthyroidism has no such limited distribution. Those of us, therefore, who are outside of the goiter districts, should bear the possibility of this condition well in mind, in view of the dangers to be considered later, which may arise from illadvised procedures upon patients with hyperthyroidism. The same caution applies also in the case of children. We have been apt to consider that hyperthyroidism is a disease of the later years of adolescence and of early adult life. Its incidence in young children has been generally supposed to be so rare that it is hardly to be considered. In the Cleveland Clinic, however, forty cases of hyperthyroidism have been seen in children under fourteen years of age, and various groups of cases in children have been reported in the literature at varying ages under fourteen years of age; one being only nine months old. In our series the ages varied from five and one-half to fourteen years of age, and in one case it appeared from the history that the disease was initiated when the child was but three years of age. Although this is not a high incidence as compared with the cases of hyperthyroidism encountered in later years, it is a sufficiently high incidence to warn us that we should be especially on our guard when children come to us who present a syndrome of symptoms which suggests the presence of hyperthyroidism. In general, however, the diagnosis should rarely be mistaken in such cases, the symptoms are usually outspoken in children and the characteristics of the onset are fairly uniform. Parents usually first notice increasing nervousness and irritability which is followed by a rapid enlargement of the gland, tremor and in many instances—fifty per cent in the Cleveland Clinic series—by exophthalmos.

A word regarding the pathology of the disease may be of interest particularly since it can be confined to the one statement that there is no specific pathology. There is no known change in the thyroid gland itself, or in the organism as a whole, which is pathognomonic of hyperthyroidism. The fact that there is no specific pathology is one of the reasons why the etiology and exact character of the disease is so uncertain.

As for the establishment of the diagnosis in uncertain cases, various tests have been suggested which may be made in adult cases, but are of little or no value in establishing the diagnosis in children. Among these tests are basal metabolism estimations, the Goetsch adrenalin test, the Braun quinine test, and examination of the blood. The warmest supporters of any of these methods, however, themselves emphasize the fact that these tests are of minor value as compared with the anamnesis and the clinical findings.

It is of special importance that the physician be not misled by the size of the goiter. In some cases also, in which there are no external evidences of thyroid enlargement, a goiter may be present which is extending down into the chest. These intrathoracic goiters often bring to the otolaryngologist some of his most serious problems.

The role of infection in the etiology of hyperthyroidism and goiter as indicated above is uncertain. Its role in the course of hyperthyroidism on the other hand appears to be definitely established. It may be stated here, however, that although it would appear that in general hyperthyroidism is not caused by infections, so many instances in which hyperthyroidism has immediately followed an attack of influenza have been noted that this fact may be of some etiologic significance. Aside from this one instance, however, the role of infections seems to be at present an exacerbation of the hyperactivity of the thyroid gland. This hyperactivity of the thyroid gland in turn produces an increased susceptibility to the infections, so that a vicious circle is established.

The role of infections in cases of hyperthyroidism in children is of especial importance to the otolaryngologist. In eight of our series there was a history of an antecedent infec-

tion; in one the onset of the disease appeared to be directly related to an attack of whooping cough; in another exophthalmos developed three weeks after an attack of scarlet fever. Cases are reported in the literature by Klein, in which hyperthyroidism developed immediately after tonsillectomy, and one case is reported by Wheeler in which exophthalmos followed an attack of varicella and mastoiditis; in these cases the hyperthyroidism was associated with status lymphaticus. Although the percentages are small, nevertheless the close association of the development of hyperthyroidism with the previous infection is certainly significant.

Those who believe that hyperthyroidism has a neurogenic origin cite cases in which the syndrome has immediately followed shock, fright or great emotional strain. One of our cases in the children's series immediately followed an unfortunate experience in a dentist's chair. The child went home in a state of great emotional excitement, with greatly accelerated heart action, and within a week, though kept in bed, she exhibited the characteristics of a typical hyperthyroidism syndrome, including early exophthalmos. Crotti reported a case, operated upon at Kocher's Clinic, in which the symptoms of hyperthyroidism appeared within 48 hours after the San Francisco earthquake. I believe, however, that in the cases which follow exanthemata, or surgical procedures such as tonsillectomy, an unrecognized state of hyperthyroidism already existed which was aggravated by the infection, operation, or other existing factor.

That any focus of infection should be cleared up is unquestionable. But when it should be removed is again a problem the solution of which is not easy. In cases of an intense hyperthyroidism the removal of infected tonsils, the clearing out of sinuses, the extraction of teeth, may be sufficient to kill the patient. In children, in particular, in the extraordinary hypersensitive state produced in them by the hyperactivity of the thyroid gland, mistimed operative procedures are peculiarly dangerous. In children also the presence of a thymus gland often associated with hyperthyroidism may add materially to the operative risk.

The prime problem which confronts the otolaryngologist then, in connection with hyperthyroidism, is on what basis to

judge what shall be the primary procedure, that is, whether or not the patient shall be referred to the surgeon or the roentgenologist for primary treatment of the hyperthyroidism, or whether the infection shall receive the primary treatment. The necessity for recognizing the condition in patients who come to him either of their own volition or who may be sent by physicians who have not recognized the signs of hyperthyroidism is obvious, and here the otolaryngologist should be informed regarding the relative merits of radiation and of surgery in the treatment of the disease, and of the methods employed by the internist, in order that his judgment may be based upon a thorough knowledge of the most efficient prevailing practice. For myself, although radiation therapy has many adherents whose statistics appear to justify their claims, I believe that the surgical removal of the gland is the only method whereby the hyperactivity can be directly and permanently controlled. In addition, even in an extremely mild case of hyperthyroidism in which it may appear both to the otolaryngologist and to the referring physician that removal of the focus of infection may be a sufficient measure to control the hyperactivity of the thyroid, the former should inform himself thoroughly by a physical examination and with the aid of the roentgenogram as to the relation of the gland to the trachea. It is of prime importance that the otolaryngologist know whether or not the trachea is in any degree distorted or compressed by an intrathoracic enlargement or other pathologic condition within the chest, which may increase the risk of the operation. In general, I might state that it is my own conviction, and I believe that this judgment is shared by the surgeons of the Cleveland Clinic, that any otolaryngologic operation should, if possible, be deferred until the operation has been directed to the cure of the hyperthyroidism itself. The tragic sequelae of tonsillectomies performed upon patients with hyperthyroidism illustrate dramatically the extent to which the effects of each are intensified by the other. I believe that during operations certain deaths have occurred which have been due to unrecognized hyperthyroidism, or to some mechanical condition, such as pressure upon or distention of the trachea by a goiter. In some cases the patient does not recover promptly from the anesthetic; nausea and

vomiting persist, the pulse rate mounts to an alarming degree, to 150 or 160; there is intense restlessness and delirium may develop. It is because of this dangerous sequence of events which almost invariably follows a tonsillectomy performed in the presence of hyperthyroidism that we believe that whenever possible primary attention should be directed to the cure of the hyperthyroidism.

In some cases the only operation indicated may be the ligation of the superior thyroid arteries. This is especially true in children in whom, as Dr. Dinsmore has informed me, the improvement following ligation is often far more pronounced than that which is seen in adults. But, as a rule, the control of the hyperthyroidism is only obtained after the thyroidectomy is performed, two or three months after the ligation. It should be emphasized that a tonsillectomy, or other operative procedure, should not follow the operation upon the thyroid within less than from four to six months. The otolaryngologist should also bear in mind that the surgical operation is but one step in the cure of hyperthyroidism. A complete hygienic nursery regimen is required for a period the length of which is in direct relation to the length of the period during which the disease was developing. Hay has recently called attention to the importance of a thoroughgoing detoxication in cases of hyperthyroidism by means of daily enemas of cool water, sweats and a carefully chosen diet in order to maintain as low a nitrogen index as possible. As toxic conditions are characterized by a high nitrogen index, this would seem of especial importance in the type of case which comes to the otolaryngologist in which the toxins of an infection are added to the toxins engendered by the hyperactive thyroid gland. In view of our present understanding regarding disturbances of the interrelation of the endocrine glands in this disease, it is necessary that a period for the reestablishment of the normal relation be allowed. Since the effects of an infection upon the organism are in many cases so nearly identical with the effect of hyperthyroidism, it follows that in these cases even more than the usual care should be given to the control of the hyperactive heart; to restoration and conservation of the function of the kidney; to the control of environmental conditions—that is, to the assurance that excitement and worry

are eliminated. This is peculiarly the case with the patient in whom the hyperthyroidism is complicated by the presence of an infectious process.

Even when every possible precaution has been taken, a period of adequate rest should follow a tonsillectomy. This is of the greatest importance if no operation for the thyroid has been deemed necessary, but even when a preliminary ligation or thyroidectomy has been performed, and an adequate interval has been allowed—even then the patient should be required to rest in bed for at least one week. After operations of a more serious nature an even longer period of rest in bed should be insisted upon, and it should be the practice of the otolaryngologist also to insist upon a prolonged period of rest away from school or from work, and when the patient goes home and the care of the otolaryngologist is ended, he should be referred to his own physician for a protracted period of watchful care and guidance.

In certain cases, such as an acute suppurative mastoiditis when the operation cannot be delayed, the resources of the specialist may be taxed to the utmost. For the control of such cases, he should have at his command every possible restorative and conserving measure. Acute mastoiditis in the presence of hyperthyroidism is a very menacing situation. In such a case the specialist should protect himself by insisting upon the presence of the surgeon or of the patient's own physician. In case an operation under local anesthesia is contemplated, the fact that thyroid activity is increased by adrenalin shows the importance of omitting the use of adrenalin if it is at all possible to get along without it. More sedative measures should be employed before and after operation than is the case in the usual uncomplicated operation. That is, the preoperative distress of mind and the postoperative distress of body should be reduced to the very minimum. As Crile has shown, every nervous reaction, and even the slightest degree of pain, has its effect upon the organs of the kinetic system among which the thyroid plays a major role. The less pain, the less nervous excitement, the more rest, the more sleep, by so much the more will the welfare of the patient from the immediate operation be promoted and the danger of increased hyperthyroidism be lessened. The heart should be

fortified by digitalis if there is the slightest indication of myocardial weakness. To use Crile's dictum the restorative and conservative measures should be used "in advance of the emergency." Because of the increased heart action more bleeding than usual occurs during certain operations, and there is greater danger of secondary hemorrhage; therefore, before operation the blood of the patient should always be grouped, and provision made so that a blood transfusion can be done without delay should it be necessary.

It will be noted that I have made no attempt to distinguish between hyperthyroidism and the socalled toxic adenomata. If such a distinction is of value, it does not pertain to this discussion, for as far as the otolaryngologist is concerned, his management of the case should be as outlined.

The specialist should therefore be familiar with the symptoms and causes of hyperthyroidism, and with the most efficient methods for its treatment for the following reasons:

1. The relation of infection to hyperthyroidism is not definitely known; and the removal of a focal infection is not a cure for the hyperthyroidism.

2. Under ordinary conditions, and especially in children, the surgical removal of a focal infection should be deferred until the hyperthyroidism has been controlled by surgical treatment.

3. A sudden death during an operation upon the throat, which is often attributed to the anesthetic or to cardiac failure, may be due to the presence of unrecognized hyperthyroidism.

4. Throughout the entire conduct of a case of hyperthyroidism unusual preoperative and postoperative precautions are essential.

5. When it is imperative that an operation be performed in the presence of hyperthyroidism, the presence of the internist at the operation and his cooperation during the period of convalescence is of utmost importance.

XI.

THE SIGNIFICANCE OF THE SCIENCE OF SPEECH CORRECTION.*

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ST. LOUIS.

Speech correction, handicapped and distorted by charlatans, commercialized stammering institutes and inadequately qualified teachers of voice, has at last reached the dignity of a science, due largely to the earnest and untiring efforts of such medical pioneers as Kussmaul and Gutzmann, Flatau and Avellis, Zwaardemaker and Mygind abroad, Bluemel and Blanton, Martin and Greene and our own colleagues, Kenyon and the late George Hudson Makuen in America.

From the time that Demosthenes, the silver tongued orator of Greece, cured himself of stammering by juggling handfuls of pebbles in his mouth, to almost our own era, when Dieppenbach, in 1841, proposed an operation for resection of the tongue for the radical correction of stuttering, literature and history have jumbled facts and fancies associated with these human imperfections into a chaotic mass. The stammerer and the aphasic have stumbled through the ages, beset by accusations of witchcraft and mysticism, suffering as the scapegoats of ignorance and fanaticism and ostracised from social intercourse by the mental impotence and helplessness of their fellows to devise ways and means for overcoming such defects.

As knowledge was unfolded in anatomy and physiology of the speech mechanism, in neurology and the significance of cerebral localization, in psychology and the relation of mental to physical stimuli, the fundamental principles were created for the establishment of a new science—the study, classification and correction of defects in speech.

It is not the purpose of this paper to trace the evolution of this comparatively young science through its period of struggle

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and misunderstanding. Let us rather view it today as a healthy young structure, built by sincere workers in medicine and pedagogic science of a substantial mass composed of anatomy, physiology, neurology, psychology, the physics of sound, the art of expression and the philosophy of language.

It still remains for laryngology, when convinced that correction of defects in speech has actually attained the scientific dignity which we claim for it, to adopt this young offspring of the older sciences as one of its own working forces. Here is a real and almost made to order instrument to add to the armamentarium of the laryngologist which, when well used and properly directed, will accomplish much for humanity. More than five per cent of all school children in the United States have some form of defect in speech, including stammering, stuttering, lisping, lolling, "baby talk," and various aphasias. This field of operation is numerically larger than perhaps all other activities in laryngology combined. Yet American laryngologists who have actively interested themselves in this cause are few in number. The correction of defects in speech is as legitimately a field in laryngology as are aphonias due to paralysis of the recurrens or nasal obstructions due to deflections of the septum. These speech defects are as tangible as laryngeal neoplasms and are as amenable to correction. Stammering, defined as a speech inability due to spasmodic or incoordinate action of the muscles of respiration, phonation or articulation, is largely a laryngologic problem.

We seriously study the pathology of the recurrent laryngeal nerve in tabes, but disregard the same nerve impulses when they produce a stammerer. We operate plastically and beautifully for harelip and cleft palate and discharge our obligation when our surgery has been successfully completed, overlooking the important utilitarian needs of the child to acquire motility of the palate, proper resonance and tone placement in voice production throughout his school period. We punctiliously inform the parent of a child from whom masses of adenoids and tonsils have been removed that the patient is now free of mouth breathing causes, mushiness of speech and local foci of infection, but we utterly disregard the psychology of the stammerer who has the same right to an edu-

cation and who, with his speech handicap, hesitates to recite his lessons, to speak to his fellow pupils and his friends, and whose mental and pyschologic nature constantly undergoes depressions and tortures and who, reprimanded by teacher and parent, discouraged by poor scholarship, frequently shuns the schoolroom and becomes a charge of the truancy courts.

Every larnygologist has had occasion, in his own experience, to see many cases of "baby talk." Intelligent mothers, when their attention is directed to this early defective and unintelligible form of speech, will remark, "Oh! Doctor, isn't it cute?" Instances have been brought to my attention where experienced laryngologists have replied to this remark with a graceful gesture and a look of wisdom: "Don't worry, madam, the child will outgrow it."

Last year two Czecho-Slovakian children, a girl of nine years and her younger brother of seven years, were referred to me and reported as congenitally deaf. On first examination this seemed apparent, but on more careful study it was found that these children heard practically everything that was said to them, but were unable to produce what they heard in speech. We made the diagnosis of "word deafness," a condition somewhat analogous to color blindness, and explained theoretically by the fact that the Wernicke center in the cortex (speech hearing center) and the Broca center (speech producing center) were both intact, but that the connecting tract or fibers between these centers were undeveloped or imperfectly stimulated. After one year of intensive pedagogic training similar to that applied by speech methods to the congenitally deaf child, these children have acquired a working vocabulary, speak spontaneously and, in another year or two, will be sent back to the normal schoolroom.

The more profound field of the laryngologist includes laryngectomy. With the total extirpation of the larynx, we might conclude that all possibility of speech in such a case had been forever lost. I cite a laryngectomized case referred to us by Dr. Vilray P. Blair one year after operation. Six months of special pedagogy enabled this man to speak with voice, and without any artificial means whatever, so that he could be heard across a twenty-foot room.

In the reconstruction work after the war many unusual and interesting cases came to our attention, and it is gratifying to report that a wide field of practice and usefulness was developed in the rehabilitation of soldiers who have been shell shocked, shell concussed, shrapnelled in the speech areas of the head, in the jaws, in the mouth and in the larynx, and who, by the painstaking care of well trained teachers and guidance of the laryngologist experienced in this work, can be restored to a practical and utilitarian status.

These instances should suffice to substantiate my plea that the correction of defects in speech affords even the most ambitious among us every facility for profound and dignified research and a practical application of many experiences which we have gleaned in general laryngology. This science is worthy of every support and upbuilding which laryngology can offer; its best interests have been served by a few; let the laryngologist in every community interest himself in the segregation, in the classification, in the pedagogy and in the uplift of the large group of cases of defects in speech to be found everywhere, so that laryngology may share in the glory and development of this splendid and useful young science.

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XII.

NEW METHODS AND APPARATUS FOR TESTING HEARING.*

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During the past four or five years a new interest has been taken in methods of testing hearing. The making of actual measurements is certainly the basis for progress in understanding the many difficult problems concerning hearing. Since the advent of the vacuum tube and the stimulated interest in acoustics brought about by radio broadcasting, new tools have become available, which, when given the proper form, are valuable to the otologists as measuring instruments. It is the purpose of this paper to describe some of these new tools and to show their relation to the older methods of testing hearing.

The kinds of hearing tests which are needed may be classified into four groups, according to their purpose, as follows:

1. Industrial, or those made for determining the fitness of a candidate for employment. In certain types of work, it is particularly important that a prospective employee meet a definite requirement for acuity of hearing. Tests made in the army and navy for various branches of service are conspicuous examples of this kind of test.

2. Educational, or those made for determining the degree of hearing of school children, both in the public schools and in the schools for the deaf, for the special purpose of determining the proper methods to be used in their education.

3. Clinical, or those made for assisting the doctor to make a proper diagnosis of the cause of deafness.

4. Research, or those made to determine new facts about both normal and abnormal hearing.

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*Lecture and demonstration given before the American Academy of Ophthalmology and Oto-Laryngology in Chicago, October 21, 1925.

It is evident that different methods are appropriate for these different purposes. Unless these various purposes are kept in mind, there is apt to be confusion when discussing the merits of various methods of testing hearing.

It is highly desirable that a single scale be used for representing the degree of hearing, which is independent of the method used, and which has a general application to all the purposes mentioned above. Therefore, before describing the apparatus developed for making hearing tests for these various purposes, I desire to describe the scale of hearing which is now being used by a number of otologists, which may be used for all these purposes, and show how the results obtained from the commonly made voice test, watch tick, acoumeter, coin click, and tuning fork tests can be converted into hearing loss units on this scale. This scale for measuring hearing was first described in a paper given before the Franklin Institute in September, 1923. It is the one used on the audiometers manufactured by the Western Electric Company. However, the use of this scale is in no way dependent on the use of audiometers of this kind. The units have been found to be very practical for describing the amount of hearing, since one of the units corresponds to a fractional change in the intensity of a sound which is approximately the smallest that the average ear can detect. For example, when one changes the intensity of his voice from that corresponding to the musical notation PP (pianissimo) to that corresponding to the musical notation MF (mezzoforte), the loudness change is about 15 of these units. There are about 15 loudness steps in this range that the ear can recognize as being different. The range of intensities, from the threshold of audition to the maximum which the ear can tolerate, is approximately 130 of these units, depending upon the kind of sound. The hearing loss is defined accurately by the equation

$$\text{H. L.} = 10 \log \frac{I}{I_0}$$

where I and I_0 are the intensities necessary for the sound to be just audible to the person being tested and to a person of normal hearing, respectively.

It is not necessary, however, to remember this formula or even understand its significance, to be able to use in a practical way the scale of hearing just described.

In a paper presented before the American Triological Society by Fowler and Wegel (Audiometric Methods and Their Applications, May, 1922), a hearing scale was proposed which has been objected to by some otologists because it is dependent upon the threshold of feeling as well as the threshold of hearing. On this scale the per cent hearing loss is the number of sensation units from the normal to the threshold for the patient divided by the number of sensation units from the normal to the feeling point for a person of normal hearing. It is undoubtedly the best answer to the practical question as to what is the per cent hearing loss, and is very useful in expressing general results. It is particularly useful for describing to the patient his degree of hearing. However, for an accurate expression of the degree of hearing loss, it seems desirable to express results in terms of sensation units rather than per cent hearing loss.

The hearing loss expressed in sensation units can be converted into per cent hearing loss by multiplying the former by a factor K which varies with the type of sound used in the test. It is obvious from the definitions given above that this factor is equal to 100 divided by the number of sensation units between the threshold of hearing and the threshold of feeling for the particular sound used. Experiments made in the Bell Telephone Laboratories have indicated that for speech this factor is .83; for the test tone of 3-A audiometer it is equal to 1.0; for a watch tick, coin click, or acoumeter it is approximately 1.5. For pure tones the value of K is dependent upon the pitch and is given in Table I. The question marks after 32 and 48 indicate that these values are not accurately determined. By means of this table, the hearing loss in sensation units can readily be converted into per cent hearing loss. For example, if a test indicates that a person's hearing is 50 units down from normal, his per cent hearing loss is $50 \times .83$ per cent, or 41.5 per cent.

TABLE I.

FACTORS FOR CONVERTING HEARING LOSS EXPRESSED IN SENSATION UNITS INTO PER CENT HEARING LOSS

Vibration Frequency	K	Vibration Frequency	K
32	3.3 (?)	800	.77
48	2.2 (?)	1024	.76
64	1.5	1500	.77
100	1.28	2048	.77
128	1.09	3200	.81
200	.96	4096	.86
256	.91	6400	.94
400	.83	8192	1.22
512	.79	12800	1.42
Speech or 4-A Audiometer			.83
Test Tone (3-A Audiometer)			1.00
Watch Tick or Acoumeter			1.5

When speech tests, as ordinarily made by otologists, are made in rooms with highly reflecting walls or in a place where there is noise, it is impossible to reduce the results so that they may be compared with other results because of so many uncertain factors. However, if the tests are made in a room which is heavily damped and quiet, the distance that numbers can be interpreted correctly is definitely related to the hearing loss on this new scale. In Table II are given numbers which show this relationship for four different intensities of voice. The derivation of these figures is based on some experimental data obtained in the laboratory and from the inverse square law relating to the intensity of sound and the distance from the source.

TABLE II.

ROOM HEAVILY DAMPED AND FREE FROM NOISE

Distance in Feet	Hearing Loss in Sensation Units			
	Average Whisper	Loud Whisper or PP Voice	MF Voice	FF Voice
⅛	50	65	80	95
¼	44	59	74	89
½	38	53	68	83
1	32	47	62	77
2	26	41	56	71
4	20	35	50	65
8	14	29	44	59
16	8	23	38	53
32	2	17	32	47
40	0			
64		11	26	41
128		5	20	35
226		0		
248			14	29
496			8	23
992			2	17
1280			0	
1984				11
3968				5
1⅓ miles				0

Only very approximate results can be obtained by such speech tests, since it is very difficult to determine the particular type of voice one uses in calling numbers. It is interesting to know that when one is using a very loud voice corresponding to shouting, one can hear and interpret called numbers at a distance of 1⅓ miles, provided, of course, that everything is perfectly quiet. A person with normal hearing can also hear and interpret numbers which are whispered at a distance of 40 feet when everything is perfectly quiet. For example, if one is using an average tone of voice in a room which is heavily damped and free from noise, and finds that the patient can interpret his numbers at a distance of four feet, the hearing loss would be 50 sensation units. In most rooms in large cities, unless precaution has been taken to eliminate the noise, one will obtain distances corresponding to between 20 and 30 units loss in hearing for a person having normal hearing. It is also seen from the figures in Table II that it is not practical to determine the loudness of one's voice by noting the distance that the normal ear can hear, since these distances are

all too large to be obtained in an ordinary sized room. If distances smaller than these are obtained it indicates that the noise has not been excluded.

This same method can be used for reducing watch tick or acoumeter results to the common scale, provided the intensity of the watch tick or acoumeter is known. A number of observations made with good grade watches indicated that a patient who could just hear a watch tick which was held on the ear has a 50 per cent hearing loss in the neighborhood of 2,000 cycles.

In the standard method for determining the acuity of hearing at different pitches use has been made of a series of tuning forks having various vibration rates through the audible range. The fork is given a standard blow of some sort to set it into vibration. It is then held as close to the ear as possible, preferably with the flat part of the prong directly facing the auditory meatus. The time t in seconds from the striking of the blow until the patient no longer hears the sound is observed. A comparison of this time to that t_0 required for normal hearing gives a measure of the hearing by air conduction.

It is well known in mechanics that after a tuning fork is set into vibration the difference in the logarithms of the amplitudes of the sound emitted by the fork at two different times is proportional to the difference in the two corresponding times elapsed from the time of the initial blow.

Due to this relation and the way that hearing loss is defined on the new scale, it is easy to show that the hearing loss is equal to a constant designated by Δ and called the damping constant, multiplied by the difference in time t_0, for the normal ear, and t for the ear of the patient being tested. The constant factor for reducing the time difference to sensation units is only dependent upon the fork and should be furnished by the manufacturer.

For illustrating the kind of constants one might expect to obtain the values of Δ for, three groups of tuning forks are given in Table III. For convenience, the values of K from Table I are also given. The first group of forks are from a set used in the Bell Telephone Laboratories. The second set was loaned to us by Dr. E. P. Fowler. The last fork is a 500 cycle standard fork used by Dr. Douglas MacFarlan.

For example, when Dr. Fowler's 256 fork is used, the per cent hearing loss is given by

$$\text{Per cent hearing loss} = 1.25\ (70 - t)$$

where t is the air conduction time for the patient. It is evident from the formula that a person having more than 87½ per cent loss will not hear the fork at all.

It is useful to notice that if the hearing loss of the tester is known, then the hearing loss of the patient can be found as follows: Set the fork into vibration by any means, not necessarily using a standard blow. Hold the fork to the patient's ear in the standard fashion. Start the stop watch when the patient signals he no longer hears the tone. Hold the fork to your own ear in the same standard way. Stop the watch when you cease to hear the tone. Then the reading of the watch in seconds, multiplied by the constant of the fork Δ, gives the difference in sensation units between your hearing loss and that of the patient.

TABLE III.

TYPICAL CONSTANTS FOR TUNING FORKS

Rate in dv	Damping Constant Δ	Time t_0 in seconds for Normal Air Conduction	Factor K for Reducting Hearing Loss to % Hearing
	Bell Telephone Laboratory Forks		
24	.30	75	4. (?)
48	1.9	51	2.2 (?)
64	1.61	41	1.5
100	1.75	30	1.28
200	.93	110	.96
400	.46	140	.83
500	.59	135	.81
800	.87	112	.77
1000	1.19	71	.76
1200	1.14	69	.76
1800	2.29	44	.77
2000	2.41	45	.77
	Dr. E. P. Fowler's Forks		
128	1.08	65	1.09
256	1.38	70	.91
512	1.31	95	.79
1024	1.70	40	.76
2048	2.17	20	.77
	Dr. D. MacFarlan's Standard 500 Cycle Fork		
500	2.0	55	.81

$$\text{Hearing Loss} = \Delta\ (t_0 - t)$$

$$\text{Per Cent Hearing Loss} = K\ \Delta\ (t_0 - t)$$

This method has the advantage that the results are not dependent upon the initial blow given to the fork, but it has the disadvantage that it depends upon the hearing of the tester, and any noise in the room affects the hearing of the tester much more than that of the patient, provided the former is much more acute than the latter.

For example, suppose that you know that the tester's hearing loss is 20 SU at 2,048 vibrations per second, and also, when using the technic described above and Dr. Fowler's 1,024 fork, the time difference is found to be 25 seconds. Then the hearing loss of the patient is

$$1.70 \times 25 + 20 = 62.5$$

or his per cent hearing loss is

$$62.5 \times .76 = 47.5 \text{ per cent}$$

It would be a great advantage to those who use tuning forks if the makers of such forks for otologic purposes would furnish values of the damping constants as well as the frequency of vibration, then the results of tuning fork tests could be reduced to a common basis, as indicated above.

In order to increase the speed and accuracy of making hearing tests which are equivalent to those which have been described and for the purposes outlined at the beginning of the paper, the Bell Telephone Laboratories have developed several pieces of apparatus which I wish to present for your consideration. For making speech tests a phonograph type of audiometer has been developed. In this instrument records which have been made by special electrical processes are used for reproducing the speech sounds. This insures a definite volume of voice, so that accurate comparisons are possible. The recorded speech sounds are transformed into their electrical equivalents by means of an electromagnetic reproducer. The electrical waves thus created are carried by means of wires to a receiver which is held to the patient's ear. The intensity of the sound sent into the patient's ear is controlled by electrical resistances in the electrical circuit. This phonograph type audiometer is furnished in three different forms, according to the purpose for which it is to be used. For making a survey of the hearing of a large group to find those who have a deficiency for hearing speech, it is furnished with the turntable

unit, with special records upon which the recorded speech sounds continually decrease in intensity as the record revolves, and multiple units containing eight receivers per unit. A master sheet is furnished, which gives the hearing loss corresponding to the intensity of each of the numbers on the record. In this form no batteries or attenuators are required. An instrument of this sort was tried out at one of the public schools in New York City, and it was found that with a single phonograph unit and three multiple units—that is, 24 receivers—the pupils could be tested at the rate of 100 per hour. The entire school of about 1,000 pupils was tested in three days, and a number determined for each pupil, which represented his hearing ability for speech. (Demonstrate use of this instrument by connecting it to the public address system and attenuating until a person in the front of the hall hears the numbers corresponding to normal.)

For purposes of testing more accurately the hearing of one individual at a time, an attenuator is interposed between the turntable unit and the receiver, and a record is used which gives a constant speech level. For use in schools for the deaf or for testing patients who are very hard of hearing, an amplifier unit can be furnished, to be interposed between the turntable unit and the attenuator box. (Demonstrate the use of this instrument with a 25-A amplifier.) This phonograph type of audiometer can also be used as an auxiliary to either the 1-A, 2-A or 3-A audiometer. When used in this way the hearing loss can be read directly on the audiometer dial. (Demonstrate its use on both the 1-A and 2-A audiometer.) With this instrument the speech intensities being delivered to the ear of the patient can be varied 10 billion fold by simply turning the dial from one end of the scale to the other, corresponding to a distance variation of more than 100,000. This makes it possible to test all degrees of hearing from normal to those who are totally deaf. Not only does it have this increased range over that obtainable in the ordinary speech test, but it has a definite intensity level for the speech and is not subject to variations in the acoustic properties of the room.

The 3-A audiometer, which has been described before, has been developed to take the place of such tests as the watch tick tests, the acoumeter tests, or those designed to make a very

quick test of the general hearing level. In this instrument a tone having components throughout the entire speech frequency range is electrically generated and delivered to a receiver to be held on the ear of the patient. The volume of the tone is controlled by the same attenuator unit as used in the other audiometers. It reads directly, either in sensation units loss or per cent hearing loss, since it was found for this tone that there were just 100 SU between the threshold of hearing and the threshold of feeling for the normal ear. This instrument has been found to be particularly useful in schools for the deaf. It enables the teachers to grade the degree of hearing of the child very quickly, which aids them in deciding upon the kind of methods to be used in teaching him. It is also useful in making a quick test of the hearing of large groups when they are tested one at a time. (Demonstrate the performance of the 3-A audiometer.) This 3-A audiometer is also useful when making tests of bilateral deafness, for it can be used in place of the Bárány noise apparatus. For this purpose it is much superior to the latter device, because the volume can be placed at just the proper level to sufficiently mask the hearing in the good ear without in any way interfering with the hearing in the bad ear. The proper level for doing this depends upon the relative difference in the hearing of the two ears. Those who believe an ear massage is of value may find an additional use for this instrument.

The apparatus which has been developed for replacing the tuning fork test is now quite well known. Several types of audiometers have been developed for this purpose. The two which have been developed by the Bell Telephone Laboratories and known as the 1-A and 2-A audiometers are here for demonstration. The 2-A audiometer has been designed for general practice, and for this reason great stress has been placed on the convenience and simplicity of its operation. Eight tones spaced an octave apart in the frequency range, from 64 to 8192 vibrations per second, can be produced by throwing the appropriate keys on the top of the audiometer. The attenuator scale is calibrated so that the hearing loss in sensation units is read directly from the scale when the dial is turned to the position corresponding to the threshold for the patient being tested. Audiogram blanks are furnished with the instru-

ment, which make it possible to show graphically the hearing loss at each frequency. Already a number of doctors have changed the form of these blanks to fit their particular need, but, of course, have retained the same scale.

The 1-A audiometer is similar in principle to the 2-A audiometer, but it has a much greater range in frequency and intensity. Both of these instruments are equipped with a jack, so that auxiliary equipment may be used with them. It has been our experience that the doctor frequently desires to talk to the patient either before or during the time that the pitch-range test is being made. For this purpose a talking set has been developed. By plugging the end of the cord of the talking set into the jack of the audiometer, it is possible to talk directly to the patient through the receiver which he is using during the test. By turning the attenuating dial, the volume of the speech thus delivered to the patient's ear can be varied in intensity from high values to the threshold of audibility. (Demonstrate the use of this instrument on both types of audiometers.) Also, as mentioned before, the phonograph audiometer may be used as an auxiliary to both the 1-A and 2-A audiometers by plugging the cord from the turntable unit of the phonograph audiometer into the appropriate jack. Then the speech waves coming from the phonograph record will be delivered directly to the receiver of the patient's ear. Its volume may be controlled by the attenuator dial. A mark on this dial can be determined so that readings on the scale from this mark will give directly the patient's hearing loss for speech.

In schools for the deaf, and other places where large intensities are required and where the expense of a 1-A audiometer may not be justified, the intensities of the 2-A audiometer may be increased by adding the amplifier unit used in connection with the phonograph audiometer. This amplifier can be used with the 2-A audiometer by a modification in the present type. (Demonstrate the use of the 25-A amplifier with the 2-A audiometer.)

For research purposes any or all of this apparatus might be used. It may be modified in a great variety of ways to suit the investigator. For example, three different types of tone range audiometers have been discussed before this society, in which a continuous variation in pitch was possible. It has been

our opinion that, except for special research work, this is not necessary or even desirable. However, for those who like the other features of the 1-A audiometer and still want a continuous variation in frequency, I will state that a 7-A Western Electric condenser box can be connected into the circuit so that all frequencies besides those normally obtained can be produced. When the audiometer is thus converted, a calibration curve is required for interpreting the results.

In order to show comparative results obtained by different methods of testing, Table IV is given. It shows the results of tests on the hearing of ten persons by four different methods, namely, the phonograph audiometer, the 3-A audiometer, the standard speech test, and the watch tick test methods. Audiograms obtained by the 2-A and 1-A audiometers are given for these same people in Fig. 1. On these audiogram charts the hearing loss for speech and for the 3-A audiometer tone are shown at the left and right respectively. The black rectangle represents the watch tick results. A comparison of the results shown in columns III and IV shows that the phonograph audiometer gives results which are in good agreement with the reduced results from the standard speech tests. The differences are well within the observational error.

The watch tick results are just what one would expect from the amplitude frequency characteristic of this type of sound. It is interesting to note that if patients 3 and 5 were given a watch tick test only, they would be considered to have the same amount of hearing, but further tests showed that patient 3 could hear speech at 20 feet, while patient 5 could hear it only 2 feet away. It is evident from the audiograms why this is true. Patient 5 hears the high frequencies very much better than the low frequencies. An examination of the various audiogram charts indicates that the 3-A audiometer really gives a good criterion of the general hearing level. However, results obtained by it are not definitely correlated to the hearing loss for speech, although there is a general agreement. A notable exception is the lack of agreement in the results obtained by patient 10. The 3-A gave 45 SU for the right and 60 SU for the left ear. The phonograph audiometer gave the hearing loss for speech as 70 SU for the right and 75 SU for the left ear. An examination of the audiogram for patient 10 shows why this

TABLE IV.

I	II		III		IV	V		VI	VII
	Distances for Interpreting Numbers					Distances for Watch Tick Test			
Ear Number	Voice A Y=58	Voice B Y_0=65	Hearing Loss Cal. from II		Hearing Loss Phonograph Audiometer	A (Y_0=10)	B (Y_0=15)	Hearing Loss Cal. from V	Hearing Loss 3-A Audiometer
1—R	800 ft. (cal.)	1800 ft. (cal.)	0	0	0	40 in.	70 in.	0	1	0
1—L	800 ft. (cal.)	1800 ft. (cal.)	0	0	0	20 in.	36 in.	6	5	0
2—R	140 ft. (cal.)	320 ft. (cal.)	15	15	15	10 in.	31 in.	12	7	10
2—L	140 ft. (cal.)	320 ft. (cal.)	15	15	15	15 in.	64 in.	8	2	10
3—R	20 ft.	20+ft.	32	?	30	3 in.	5½ in.	22	22	30
4—L	3 ft.	6 ft.	48	49	35	—	1 in.	—	39	35
5—L	1.7 ft.	3.7 ft.	53	54	45	2 in.	4.5 in.	26	23	40
5—R	1.8 ft.	3.7 ft.	52	54	50	3 in.	6.5 in.	22	21	45
6—L	2 ft.	3.5 ft.	52	54	50	—	contact		55	45
6—R	1.5 ft.	4 ft.	54	53	45	—	.5 in.		45	45
7—R	.9 ft.	2 ft.	57	52	50	—	contact		55	45
8—L	1.2 ft.	3.7 ft.	56	54	60	—	——			50
8—R	1.2 ft.	3.7 ft.	60	54	60	—	contact		55	50
4—R	No test				65	—	——			65
9—L	8 in.	13 in.	62	64	65	—	——			60
9—R	5 in.	8.5 in.	65	68	70	—	——			60
7—L	8 in.	15 in.	61	63	70	—	——			65
10—R	3.5 in.	9 in.	69	68	70	—	——			45
10—L	2 in.	6 in.	74	71	75	—	——			60
—R	No test				90	—	——			80

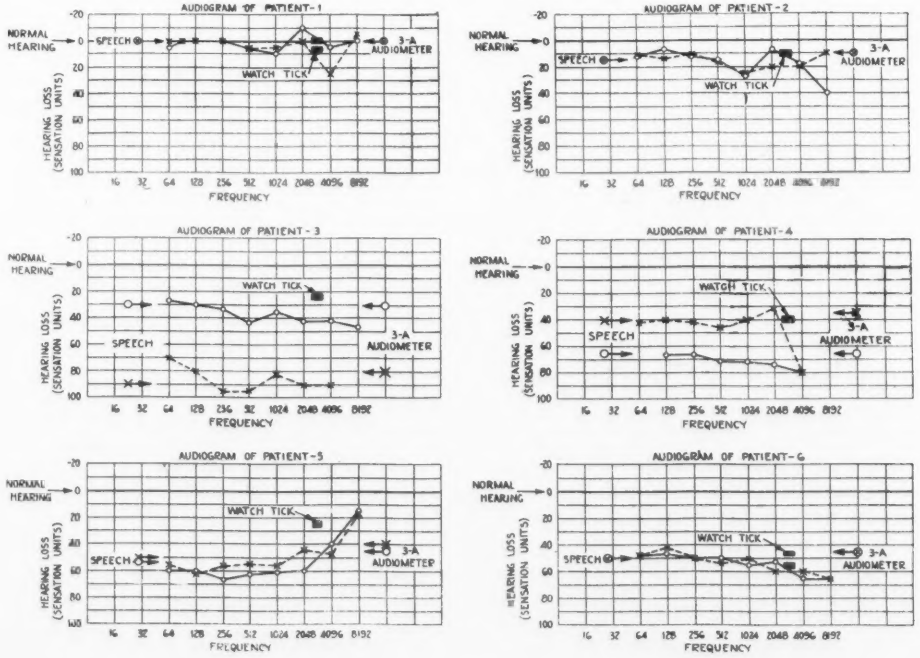
should be. The important frequencies for speech interpretation—that is, from 500 to 2,000—are considerably below the general level, the low frequencies being the highest. This is also true for patients 8 and 9. For patient 5 the speech frequencies are on about the same level as the low frequencies, but very little loss is shown for the high frequencies. In this case it is probable that the 3-A audiometer tone was heard because of components between 2,000 and 8,000 vibrations per second.

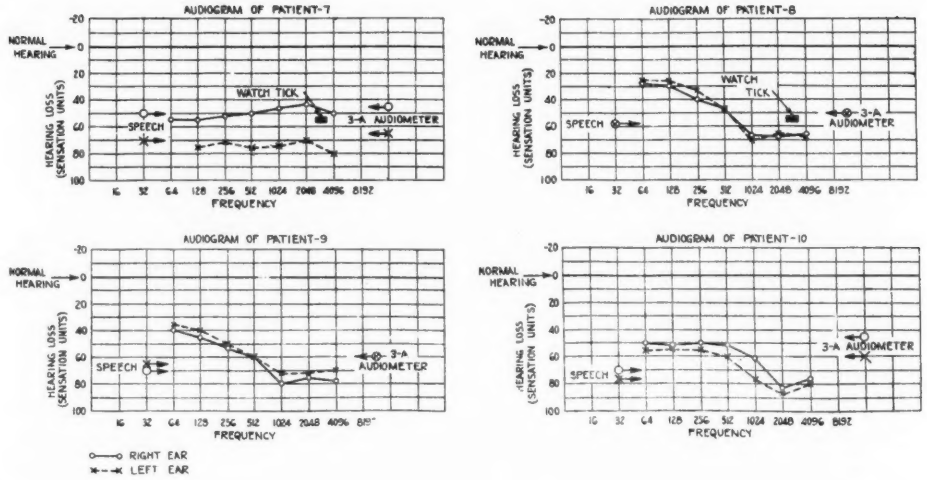
Our studies on speech and hearing have indicated that the important frequencies for interpreting speech are between 500 and 2,000 vibrations per second. The simple method of taking an average of the hearing losses at 512, 1024, and 2048 for determining the hearing loss for speech gives results in good agreement with observations on these ten patients. In Table V is shown a comparison of results thus obtained. The observed values are averages of the figures given in columns III and IV of Table IV. The per cent hearing loss is obtained by multiplying the figures by .83. Until more accurate methods are devised, this simple procedure will be useful in calculating from the audiogram the per cent hearing loss for speech which is the figure of greatest interest to the patient.

TABLE V.

HEARING LOSS FOR SPEECH

Ear Number	Calculated from Audiograms	Observed Average
1—L	2	0
1—R	2	0
2—L	20	15
2—R	16	15
3—L	39	31
3—R	89	90
4—L	40	40
4—R	72	65
5—L	52	50
5—R	61	55
6—L	55	50
6—R	48	50
7—L	48	50
7—R	73	70
8—L	61	58
8—R	60	58
9—L	68	65
9—R	72	70
10—L	75	75
10—R	66	70


AUDIOGRAM OF PATIENT-1
NORMAL HEARING
SPEECH
WATCH TICK
3-A AUDIOMETER
HEARING LOSS (SENSATION UNITS)
FREQUENCY
AUDIOGRAM OF PATIENT-2
AUDIOGRAM OF PATIENT-3
AUDIOGRAM OF PATIENT-4
AUDIOGRAM OF PATIENT-5
AUDIOGRAM OF PATIENT-6


AUDIOGRAM OF PATIENT-7
AUDIOGRAM OF PATIENT-8
AUDIOGRAM OF PATIENT-9
AUDIOGRAM OF PATIENT-10
NORMAL HEARING
-20
0
20
40
60
80
100
HEARING LOSS (SENSATION UNITS)
WATCH TICK
SPEECH
3-A AUDIOMETER
16 32 64 128 256 512 1024 2048 4096 8192
FREQUENCY
RIGHT EAR
LEFT EAR

XIII.

MASTOIDITIS IN DIABETES COMPLICATED WITH DIABETIC FACIAL PARALYSIS.

By E. M. Seydell, M. D.,

Wichita.

With the exception of mastoiditis complicating diabetes, there seems to be nothing in the literature relative to the subject to be presented in this paper. The textbooks at my disposal were singularly devoid of information on the subject. My study, therefore, was prompted by two cases where mastoiditis occurred in patients who were diabetics and in whom existed a further complication, a diabetic peripheral facial paralysis.

Diabetes is a disease noted for its numerous complications. In this disease almost any organ may become the seat of some pathologic process. The pathologic conditions, generally speaking, are not characteristic of diabetes but may occur in other diseases.

Considerable discussion has arisen relative to the causative factors of these numerous complications, especially since they represent inflammatory changes—changes due to nutritional disturbances, necrosis and degenerations.

The pathologic factors favored by the majority may be enumerated as follows: Nutritional disturbances, increase of sugar in the blood and tissues, toxic substances elaborated in the system, and changes in the vessels.

Those who favor the theory of improper nourishment point out that the resistance of the individual is reduced to a place where it is no longer possible for the organism to defend itself from the harmful influences of such factors as trauma, overexertion and infection.

The second theory which is favored, among others, by Zeimssen,[1] Naunyn[2] and Grube,[3] believe that the tissues are injured by the circulation of an abnormal amount of sugar. The injury in this instance is due, in part, to the effect of the sugar itself, as in neuritis, cataract, retinitis, etc., and, secondly,

to the existing anhydremia. From another angle, experimental work on dogs has shown that the sugar laden tissues offer a good culture media for pathologic organisms.

That the vague pains, neuralgias and neuritis bear a relationship to the sugar in the urine is shown by the fact that in early cases these symptoms clear up very rapidly after the patient becomes sugar free. In those cases where the neuralgia or neuritis has existed for a considerable time, recovery may be prolonged. The same may be said of diabetic paralysis.

Eichhorst,[4] Auche[5] and Lugaro[6] and others believe that toxins are elaborated in the system, due to faulty metabolism. A number of authors, among whom are Naunyn[7] and Partsch,[8] believe that diabetic neuritis, gangrene and other conditions are sometimes directly due to changes in the vessels (endarteritis).

Von Noorden,[9] Osler[10] and others have noted that arteriosclerosis may be caused, or increased by, diabetes. According to the histologic investigations of Hildebrand,[11] diabetes leads early to a degeneration of the contractile vascular walls, with a secondary prolification of connective tissue.

In discussing the ear complications of diabetes I shall refer to only those which relate to the subject of the paper.

According to the statistics of Koerner,[12] Eulenstein,[13] Grunert,[14] Grossman[15] and others, diabetics have a greater tendency to suppuration of the middle ear and temporal bone than nondiabetics, and acute otitis media in this malady leads more frequently to disease of the mastoid, with extensive caries and necrosis, than in nondiabetics.

Toynbee,[16] in 1860, and, later, Kirschner[17] described great destruction of the mastoid in this disease.

Wolf,[18] in 1887, was first to describe what he termed otitis media diabeticum with necrosis of the mastoid. Raynaud,[19] Walb,[20] Koerner[21] and Wild[22] even contended that the process began as a primary mastoiditis. Koerner[21] stated that diabetic mastoiditis was characterized by extensive central destruction of the bone in the mastoid process. This destruction might occur without tenderness to touch or swelling on the external surface. He considered the infection as hematogenous in origin, extending very early to the pneumatic spaces, and discharging its pus through the middle ear. Thus the entire mas-

toid might be converted into a large cavity filled with granulation tissue and pus, and covered by only a thin cortex.

Severe destruction of the mastoid is not found in all cases of diabetic mastoiditis, as was pointed out long ago by Breiger[22] and Eulenstein.[13]

The following mastoid complications have been reported: Necrosis with formation of sequestra, Massier,[23] Koenigsbauer[24] and others; sinus thrombosis, Leidler[25]; brain abscess, Heimann[26]; periarticular abscess of the maxillary joint, Ephraim[27]; paralysis of the abducens, Luc[28]; and facial nerves, the last reported by Castex[29] (the case being an old diabetic, who had a chronic suppurative mastoiditis, with facial paralysis, otitic in origin).

COMPLICATIONS OF THE NERVOUS SYSTEM IN DIABETES.

It has been proved without question that the nervous system, and, especially, the peripheral nerves, are very frequently involved in diabetes. Von Noorden[31] came to the conclusion that all diabetics have some involvement of the nervous system. Zeimssen,[30] in 1885, was the first to note neuralgias in diabetes. Von Noorden,[31] Hartman and Schrottenbach[32] and Futcher[33] find that the most frequent nerve complication in diabetes is neuritis.

The nervous complications may be divided into:

First—Sensory changes: Among the sensory changes we find paresthesia, hyperesthesia and anesthesia, neuralgia, myalgia and vague pains. These last are very frequent in the early forms of the disease.

Second—Motor paresis and paralysis: Von Noorden,[31] Bernhardt,[34] Naunyn,[35] Schmidt-Rimpler,[36] Fuchs[37] and others have found that the crural nerve is the one most frequently involved in diabetes, the abducens ranking first among the cranial nerves. Paralysis of the oculomotor, hypoglossus, spinal accessory and facial nerves have been reported in the literature.

Third—Trophic disturbances: Trophic disturbances, such as dryness of the skin, falling out of the hair, nails, etc., are frequently found.

Fourth—Loss of the tendon reflexes: Loss of the tendon reflexes was first noticed by Bouchard[37] in 1882. The statistics of Auerbach,[39] Eichhorst,[40] Neviere[41] and Williamson[42] show loss of tendon reflexes which varied from 25 to 50 per cent.

Fifth—Vasomotor disturbances may be present, as localized edema; and asphyxia has been caused by this condition.

Sixth—The special senses: The senses of smell and taste are rarely involved. Von Noorden[31] has seen six cases in late severe diabetes where the sense of taste was involved. He has also noticed two cases with parageusia.

DIABETIC FACIAL PARALYSIS.

In my study of this complication I could find only twenty cases reported in the literature: Dreyfous,[43] Gaitz,[44] L. Bruns,[45] Neuman,[46] Bernhardt,[47] Hirschberg,[48] Hatscheck,[49] Dufour,[50] Blanc,[51] Despaige,[52] Despagnet,[53] Vergely,[54] Gregory,[55] Auche,[56] LeCorche.[57] I have no doubt, however, that the condition occurs more frequently than this would indicate. There is nothing characteristic about this type of facial paralysis, excepting perhaps the pain, which often precedes and accompanies it, and is frequently quite severe, probably more so than in facial paralysis of any other origin. This pain may be present for as long as two weeks prior to the appearance of facial paralysis. A few cases have been reported without pain.

The paralysis is usually benign in character, only one case being reported in which a permanent paralysis followed. The mild cases respond very rapidly to antidiabetic treatment. The long standing and severe cases require a considerable period of time for recovery, varying from a few weeks to six months.

Failure to continue the proper diet may cause a recurrence either on the same or on the opposite side of the face. In the cases that were reported in the literature I found two were bilateral: Gregory,[55] Blanc.[51] In addition to the bilateral facial palsy, one of the patients also had an involvement of the third, sixth and a bilateral twelfth.

INTERNAL EAR COMPLICATIONS.

Greisinger was probably the first to call attention to the possibility of internal ear complications in diabetes, in 1859, by reporting three cases of partial nerve deafness coming long after diabetic eye findings were noted.

In 1905, Whitmaak,[58] Bruhl,[59] in 1912, and since then Gruber,[60] Alexander,[61] Hegener,[62] Sendziak,[63] Heiman[64] and Lang[67] have reported isolated cases. Edgar[65] seems to have been the first to have made a systematic study of this condi-

tion. He examined and observed fifty-two diabetics in the clinics of von Noorden, Ortner and Chovstek in Vienna, and found inner ear trouble in 48 per cent of these cases. His conclusion, after observing these patients, before and after treatments, and noting the frequency with which treatments were followed by improvement of the ear symptoms, was that diabetes is an etiologic factor in nerve deafness, the causation factor being a direct toxic influence (toxic neurolabyrinthitis). In other cases arteriosclerotic changes seemed to have been the causative factor, or a combination of both acting together.

The vestibular nerve is also involved in the process, as shown by the case reports of Hegener.[66] Lang[67] and six cases of Edgar's series.

PATHOLOGY OF NERVOUS CHANGES IN DIABETES.

Hartmann and Schrottenbach[68] found a degenerative neuritis with degenerative atrophy of the ganglion cells of the anterior horn in the lumbar region in a case of diabetic neuritis of the tibialis anticus. Eichhorst[69] and Auche[70] also found the same changes.

Stensay found circumscribed areas of swelling of the nerve fibers and atrophy of the medullary sheaths in both the facial and spinal accessory nerves under the same conditions. Findlay and Lugaro[71] made similar findings.

Whitmaak[72] and Steinbrugge[73] seem to have been the only authors who have reported the results of a pathologic examination of the auditory apparatus in diabetics.

In Whitmaak's case there was a degenerative neuritis of the cochlear nerve, with a normal static labyrinth, in a child who died in diabetic coma. There had been no clinical examination before death, but previous to this the attending physician noticed a moderate degree of deafness.

In Steinbrugge's case the patient had a hemorrhagic otitis media with microscopically visible extravasations of blood in the labyrinth, at the periosteum of the internal meatus, and between the fibers of the auditory nerve, etc.

A RESUME OF THE PATHOLOGIC FINDINGS.

The small number of histologic examinations made thus far does not allow one to come to any definite conclusion relative to the findings in diabetic neuritis. It is certain, however, that in many cases examined, a degenerative neuritis is present,

with atrophy of the medullary sheaths and, at times, hemorrhages.

CASE REPORTS.

Case No. 1.—Mrs. A., white, married, age 58. December 3, 1923. Chief complaint: Pain in right ear and right side of head. Present illness: The patient developed a pain in her right ear eleven days previous to date, which was continuous and throbbing in character, more severe at night, making sleep impossible. She also had pain in her gums, mastoid and temple on the right side. There was no discharge, but she noticed that her hearing, which had been somewhat impaired, became much worse during this illness. The patient also complained of vertigo. She had noticed a drooping of the corner of her mouth about the fifth day after the beginning of the pain. This distortion caused her to bite her cheek. Her appetite was good. She had frequency of urination.

Previous history: The patient developed a suppurative otitis media at the age of 16, resulting in a chronic discharge, which had been intermittent in character for several years. No history of previous pain over mastoid or signs of facial paralysis. She had been informed by a physician, about five years before this latest illness, that she was a diabetic, and had been on a diet since that time, but never strictly adhered to it, and for the past three or four months of this period had entirely neglected it.

General examination negative, with the exception of a complete facial paralysis on the right side, peripheral in type, and a slight deviation of the tongue to the left. The right ear: No discharge present. Drum showed a large scar but no perforation. A slight bulging was present, but the drum was only slightly injected. Tuning forks were not heard in the right ear, nor was the acumeter. Voice could be heard at concha with noise apparatus in left ear. A slight rotary nystagmus to the right (first degree) was present. Irrigating the ear with cold water produced a prompt and violent reaction. Fistula test was negative. The mastoid was not edematous or tender to pressure.

The left ear: Weber lateralized to left ear. The Rinne was positive. Moderately reduced perception for both high and

low forks. The acumeter was reduced to 18/40 feet and the voice to 22/40 feet.

Laboratory examination: Urine examination: Sugar, 4 plus; acetone, positive; diacetic acid, present; albumin, negative; blood sugar, 333 mg. per 100 c. c. of blood. White blood count, 6,500. The X-ray showed a complete sclerosis of the mastoid.

Course and treatment: A paracentesis was performed. No pus found, but after 24 hours there was moderate serous discharge. The patient was taken to a local hospital and placed under the care of an internist. Proper diet was prescribed and insulin administered. The pain continued even after the urine became sugar free. The presence of a facial paralysis, with pain in the ear and mastoid so severe that it was necessary to give opiates in order that the patient could rest, together with the ear history, made us decide on a mastoid operation. This was performed ten days later, under local anesthesia. The entire mastoid was found to be sclerotic. No pus was found.

The operation did not give the patient any relief from her pain, which, however, very slowly subsided under general treatment, but did not disappear for about two weeks. It required two months for the complete recovery of the facial paralysis. The nystagmus and vertigo disappeared early and her hearing improved.

On September 6, 1924, nine months subsequent to her first visit, the patient returned to the hospital with exactly the same symptoms experienced in the previous attack. This recurrence resulted from improper diet. The paralysis was again complete, and on the right side, and, as before, followed severe pains in the ear and side of head. In this attack the pain and facial paralysis were both relieved very promptly by antidiabetic treatment and diet. There were no ear findings at this time.

SUMMARY AND CONCLUSION.

The presence of a facial paralysis in a diabetic, preceded and accompanied by severe pain, which was not relieved by a paracentesis or a mastoid operation.

The facial paralysis and neuralgiform pain slowly disappeared upon the administration of insulin, together with

proper diet, the nerve deafness, vertigo and nystagmus being likewise relieved.

In addition, the patient had, nine months after the appearance of her initial attack, a recurrence of her facial paralysis and pain, both quickly relieved by the institution of antidiabetic treatment and diet.

Both attacks were precipitated by indiscretions in diet.

I am, therefore, led to the conclusion that we were dealing with a diabetic facial paralysis.

Case No. 2.—Mr. J. E. R., age 35, white, a restaurant keeper by occupation. January 23, 1924. Chief complaint: Severe pain in right ear and right side of the head, and also a discharge from the right ear.

Present illness: The pain in the right ear developed about eight weeks previously. Seven days after its disappearance he consulted a physician, who lanced the drum. The ear began to drain at once and continued to do so without interruption. Opening the drum relieved the pain for a few days, but this symptom subsequently returned. He suffered continuously, the pain being much more severe at night, necessitating opiates. There had been some tenderness over the right mastoid for about a month. About six and one-half weeks after the commencement of his pain, he noticed, on arising in the morning, that it was difficult for him to expectorate or to close his right eye. He complained of vertigo, especially when he changed his position quickly. He did not believe he had any fever. He had a ravenous appetite, and was constipated. He was obliged to urinate from four to five times at night. Denied venereal infection. He knew for about two years that he had diabetes mellitus, and the preceding April was in a diabetic coma for several days. His brother and one cousin on his father's side were similarly afflicted.

General examination: Negative, except that the knee and the achilles reflexes were absent. There was present a complete facial paralysis of the peripheral type. Sensory disturbances: Paresthesia, analgesia and thermoanesthesia were found below the knees. There was purulent discharge from his right ear, also slight dropping of the posterior superior canal wall. Slight edema over the mastoid with some tenderness to pressure. His hearing had been reduced to 22/40 for the voice, and the

acumeter could be heard at the concha only. The Weber was lateralized to the left. None of the forks could be heard in the right ear. The left ear was normal, with the exception of an improvement for the higher forks. No nystagmus present.

Laboratory findings: Urine, S. G. 1,032; albumin, negative; sugar, 4 plus; acetone, positive; diacetic acid, positive; blood sugar, 325 mg. per 100 c. c. of blood. White blood count, 7,500. The X-ray examination showed a cloudiness of all the mastoid cells.

Course and treatment: Patient was taken to a local hospital and put to bed with ice bag applied to his right mastoid. A suitable diet was prescribed by an internist, who also administered insulin. His ear and mastoid condition cleared up very rapidly under this treatment. The neuralgiform pains began to subside as his urine became sugar free, on the fifth day, and soon disappeared entirely. Within a month he recovered completely from his facial paralysis. The tendon reflexes likewise recovered their function.

SUMMARY AND CONCLUSION, CASE NO. 2.

Here we were dealing with a man 35 years of age, a diabetic, who failed to continue to observe a proper diet, and who had suffered for eight weeks with severe pain in his right ear, mastoid and side of head, which a paracentesis did not relieve. After six weeks of suffering, a facial paralysis developed, the pain continuing.

Physical examination revealed an acute involvement of the mastoid, complete facial paralysis, loss of tendon reflexes, and sensory changes in both limbs, with an involvement of the inner ear.

On administration of insulin, with observance of proper diet, the diseased mastoid bone, the neuralgia and facial paralysis promptly disappeared, also the vertigo. The tendon reflexes regained their normal reactions.

My conclusion is that we were dealing with a diabetic multiple neuritis affecting the facial nerve and both the motor and sensory nerves in the legs.

My experience with these cases would lead me to believe that surgical interference could be delayed or dispensed with in diabetic mastoiditis by the timely use of insulin combined with a proper diet.

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XIV.

THE TREATMENT OF THE COMMON HEAD COLD.

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The art and science of medicine is developing more and more along the line of science. One by one the causes of disease have been discovered and the scientific treatment applied, leaving to art an ever decreasing group of unconquered, and for the most part, indefinite diseases. But science has not yet conquered the common cold. Its causes are not entirely understood and the treatment is more art than science. The scope of this paper allows only the mention of the various treatments, detailed discussion of each being impossible.

Prophylaxis.—Only one means of prophylaxis, vaccines, has been assigned to me for consideration. Vaccines have been popular for more than a decade, and a great mass of experience has been gained. Concerning their usefulness some reports are negative, some enthusiastic, while the majority, admitting value, lay stress upon the frequent failure. The common cold may be due to the lighting up of a latent focus of infection, and experience would seem to show that where there is a focus of pus, as in a nasal accessory sinus, the use of vaccine before draining the sinus is of little value. Better results follow when there are no undrained areas of infection. Here we have only the patient's susceptibility to overcome.

Susceptibility is being successfully treated by vaccines in diphtheria, scarlet fever and many other diseases. To combat this susceptibility autogenous, combined with stock, mixed polyvalent vaccines, are thought to give the best results. Stock vaccines are considered efficient if the organism found is a staphylococcus.

As to choice of vaccines much has been written. Whether the bacteria should be killed chemically or by heat, whether vaccines should be fresh or may be kept a year or more, and whether they should be stored in the ice chest or at room temperature, are factors which make for varied actions. Special

culture media are considered necessary for the production of some vaccines.

The dose should be sufficient to cause a local reaction two inches in diameter. As the formation of antibodies is a tissue reaction, the location of the injections must vary to get the best effects. Some writers claim greater results when the day's dose is divided and given in several places.

The cause of many colds is unknown. Perhaps a filterable virus, from the effects of which recovery would be prompt but for the secondary invaders. It may be possible by vaccines to ward off these secondary invaders, thereby shortening the morbidity. If given early, their effect is the lessening of the infection element in hay fever.

For the relief of colds, vaccines have been used principally as a prophylactic measure. They are given also when there is little or no fever, in acute and subacute infections. Here the beginning dose should be one-half the usual quantity and the interval one or two days instead of four to seven.

Commercial vaccines are made up so that the average beginning dose is one-half c. c. and from eight to twelve doses are advised, the size of the injection being increased as tolerance is established.

The usual quantities given for the first injection are:

Streptococci	50 millions
Pneumococci	50 millions
Micrococci catarrhales	100 millions
Staphylococci aurei	100 millions
Staphylococci albi	100 millions
Influenza bacilli	100 millions

The application of vaccines to susceptibility and most of the phenomena of susceptibility and immunity are still in the experimental stage. Some investigators regard vaccines as foreign protein only, and doubt the specific action of a special vaccine. Even nonprotein substances are found to cause some of the same physiologic effects. Great care must be used lest we arrive at false conclusions from laboratory experiments or misjudge apparent effects on patients susceptible to suggestion.

d'Herelle by his observations of the phenomena of bacteriophagy has added a new chapter to the discussion of immunity.

He has observed the influence of an ultramicroscopic organism found in the filtrate of cultures which has a specific bacteriolytic action. According to d'Herelle, convalescence is brought about by the lytic action of the bacteriophage and passive immunity is the result of the presence of the bacteriophage. By the dissolution of the infecting bacteria we have set free the toxins which stimulate the body cells to the formation of antitoxin with the establishment of a more permanent immunity.

Confirmation of these observations is being carried on in many laboratories, and, while possibilities are bright for the utilizing of bacteriophagy in the treatment of infections, judgment must be withheld until experience has proved or failed to prove its value.

Aborting Colds.—Statistics concerning the aborting of colds are quite inaccurate, for there is no surety that the cold would have developed had there been no treatment. Experience, however, has convinced many that their colds have been broken up. The beginning symptoms are circulatory, and treatment to restore normal circulation, such as exercise, hot baths or hot drinks, should be given. A cathartic and quinin or Dover's powder often serve well, and a Turkish bath, followed always by a period of rest, is frequently efficient. Rest is usually necessary, as many "catch cold" only when tired and recover quickly on going to bed for a day or two.

Chlorin.—Among the newer treatments for aborting and for treatment of any stage is chlorin gas inhalation. The air inhaled should have a chlorin content close to .02 mg. per liter, or about one part of chlorin to 100,000 of air. Less than .01 mg. per liter is of little value and more than .02 mg. is irritating. Patients vary in their tolerance, and the concentration must be varied to suit. It is not difficult to keep between the above limits by the sense of smell.

For chlorinating, a small room may be used, or an apparatus delivering a stream of chlorinated air of the proper concentration. This latter has the advantage of portability as well as accuracy. When given in a chamber, the gas may be liberated from a tank of liquid chlorin. Another method is to set chlorin free from hydrochloric acid by the addition of potassium permanganate ($K\,Mn\,O_4 + 4\,H\,Cl = Mn\,O_2 + 2\,H_2\,O + K\,Cl$

+ 3 Cl). Using a bathroom of average size, five grains of potassium permanganate are added as required, usually every fifteen minutes, to a dish containing concentrated hydrochloric acid.

The amount of chlorin necessary depends upon the size and tightness of the room, as well as upon the number of persons present and the clothing worn, the furnishings and the moisture. Dampness rapidly absorbs chlorin. Treatment should last an hour, and chlorin must be added frequently to keep the concentration constant. As chlorin is heavier than air, an electric fan should be used to properly distribute the gas.

In addition to colds, all inflammatory diseases of the upper respiratory tract and some diseases of the lungs have been treated by this gas. The treatment has gained some disrepute because of the unfavorable action in asthma and some other chronic lung conditions. This should emphasize the inadvisability of placing chlorinating chambers in factories and institutions where there is no competent supervision.

Chlorin may be administered with benefit in any stage of acute or subacute coryza. When there is nasal stenosis the preliminary use of adrenalin or cocain, or both, are of service.

Several of my patients have ascribed an increase of symptoms to chlorin, but barring an occasional headache and a slight irritation of the nose and throat, I have felt in each case that the increase of symptoms was due to the normal course of the infection.

The results of this treatment in my hands are not as favorable as was hoped from the first enthusiastic reports, but are far better than some of the accounts published during the past year. It is surely conservative to state that in chlorin inhalation we have a valuable addition to our means of treating colds and, if properly supervised, one that is without danger.

Internal Medicine.—In the early stages of acute rhinitis, for a child one year old, hourly doses of two or three drops of spirits of nitrous ether are useful. When there is restlessness or cough, one or two drops of paregoric are well combined with it. Tr. belladonna, one drop every two hours, is of advantage in the stage of serous discharge, and syrup of hydriodic acid, ten drops every four hours, after the serous stage.

Among adults, aspirin and the other analgesics are useful for the relief of headache and muscular pain, but their routine use in rhinitis is unmerited. Hexamethylenamin is given by many, but I have never known it to help rhinitis, acute or chronic. Belladonna is best given in the combinations known as rhinitis, coryza or bronchitis tablets. They shorten the period of serous discharge, and, as some patients find this the only disagreeable stage, the remedies have the reputation of breaking up the attacks. Iodids are of service as soon as the discharge becomes mucoid, and calcidin, one grain tablet every hour, is a convenient form for administration.

Local Treatment.—It must not be forgotten that in early childhood a head cold is usually inflammation of the adenoids. Local treatment is best applied by the instillation of drops. Liquid petrolatum is useful as nose drops, and in many cases the addition of menthol, campor or iodin helps.

Iodi, .01; mentholis, .03; camphoris, .03; liquid albolene, 30. M. et Sig. 5 drops in each nostril two, three or four times daily as required.

Drops of 10 per cent solutions of argyrol, silvol or neosilvol have been found valuable. Neosilvol has the advantage of not staining and seems to be as efficient as the other preparations.

For cleaning the child's nose saline irrigations are useful. The treatment is routine in some hospitals. Among rhinologists the practice is usually tabooed because of the otitis media which sometimes follows, while many pediatricians believe more otitis is saved than caused.

For adults, in the early stage of acute rhinitis, a highly recommended treatment is the packing of the nostrils with 25 per cent argyrol solution on cotton, after the tissues have been shrunken by the use of cocain and adrenalin. Usually it is better to delay local treatment until nasal discharge has become mucoid.

When there is a mucopurulent discharge some cleansing treatment is advisable. Normal saline or one of the many commercial preparations may be used as a douche or by snuffing. Perhaps better for the few who can do it is the gargle which cleanses the nasopharynx and nose. While gargling, the mouth is closed and the head thrown forward and down, the nasopharynx and nose being thereby effectually cleansed. The

blowing of the nose following the introduction of fluids into the nasopharynx by any method has frequently forced infection into the eustachian tube. A good practice is to ask the patient to refrain from blowing his nose for at least two minutes. It is well also to caution him that blowing should be done by allowing one nostril to remain open while the other is held closed.

Most of us prefer spraying, which is safe and fairly efficient, while for office treatment suction through a small canula is useful. A snuff composed of equal parts of boric acid and borax provokes secretion and helps restore the normal function of the mucous membrane. Cocain may be used in selected cases, but with caution, as the benefit is not worth the risk. Adrenalin may be used where the nose cannot be ventilated. Little permanent good, however, is accomplished.

Applications to the nasal mucous membrane are sometimes irritating, even if the solution is mild and carefully applied. The painting with 25 per cent solution of argyrol, silvol or neosilvol is apparently beneficial. Later in the course of the infection menthol preparations are useful, and of these one of the best is coryfin, which may be used on a cotton tipped applicator.

Nasal ointments are useful after the serous stage, and one of the best on the market is dakol, which combines in agreeable form of menthol, eucalyptol and chlorin.

When the turbinates are swollen and the discharge is thick or scanty, considerable relief may be obtained by nasal tampons, such as 25 per cent ichthyol in glycerin. It is well also to treat the nasopharynx with applications of silver solutions or menthol preparations or by the insufflation of boric acid.

The use of a mild mentholated spray, following what is sometimes a disagreeable treatment, is comforting to the patient.

Physiotherapy.—Massage is one of the oldest and simplest methods of treatment. It is not generally prescribed by physicians and has been adopted by the cults who, operating under the names of osteopaths and chiropractors, are becoming a large factor in the treatment of disease. Massage of the nose, face and neck relieves congestion in the upper air passage,

while the same treatment applied to the back of the neck exercises a far wider vasomotor control.

Electrotherapeutics and light therapy are illustrated by a rebellious case of rhinitis which came under my observation. The cold had lasted more than three weeks with no indication of sinus involvement. The nasal discharge was mucopurulent, of average quantity, in which nonhemolytic streptococci were found. The principal complaint was nasal stenosis, which alternated with too much air space, and at times both nostrils were occluded. A high frequency electrode was introduced slowly as the inferior turbinal contracted, and was allowed to remain five minutes. This was followed by an ultraviolet applicator for three minutes. The other nostril received the same treatment. The effect was a shrinkage of the turbinals lasting four to five hours. After three days of treatment the rhinitis had improved, convalescence was established and all treatment stopped.

General Care.—Overheated houses or atmosphere impregnated with tobacco smoke, furnace gas or noxious odors retard recovery from rhinitis. Protection from drafts is advised for those who have not been hardened to withstand changes in temperature. Our patients do best in warm, moist air of uniform temperature. Cold air, especially at night, predisposes to laryngitis in cases of nasal stenosis. Where a cold has lasted several weeks or where there have been several reinfections the best treatment often is a change of scene.

The average head cold is of short duration, and comparatively few seek the physician's aid, thinking he has little to offer in addition to their own knowledge. To stimulate an interest in therapy I have laid stress on the newer treatments, feeling sure that some of these are of real value. If we can shorten the duration of the common cold, and this seems possible, we will have made a tremendous saving for our patients.

The final word should be concerning the colds we catch during treatment. One of the advantages of the use of chlorin is the freedom of the physician from close contact with the infection. Some of our patients are inconsiderate, and many quite unavoidably cough in the doctor's face. Special care or some special device should be used for protection while making local applications.

XV.

THE ATMOSPHERIC FACTOR IN THE CAUSATION OF COLDS.*

By Walter A. Wells, M. D.,

Washington.

"The breeze which gently moves the leaves, the wind which moans through the high forest is the sum total of all that has been. It is the material of all that has had existence, of those that came before us, of those that are no more and for whom we weep."

So writes a recent French biologist, de Varigny, paraphrasing in poetical language an opinion tersely expressed by a great chemist of former days, Dumas, in the words, "Living organisms are nothing more than condensed air."

The more we consider it, the more we must be convinced that air is and has always been the essential basis of the whole structure of life, the origin of its existence, the means of its maintenance and a great factor in its evolution.

The idea of the influence, as shown by Elsworth Huntington, throughout the ages past upon the development of life from its lowest forms to that of the highest civilization, carries with it the idea of a necessary adaptation of living organisms to the atmospheric environment.

The surface of our globe is everywhere surrounded by a layer of this atmosphere, which indeed penetrates the earth and the deepest recesses in the rocks, and the unfathomable depths of the sea. We live and breathe and have our being in the bottom of this great ocean of air, which, growing thinner as the distance increases, has completely disappeared at about two hundred miles. We are very sensible of discomfort because of rarefaction at a distance of five or six miles from the surface, and, at ten times or further, life without artificial aid would be impossible.

*Read at Symposium on the Common Cold at the annual meeting of the Laryngological, Rhinological and Otological Society, Atlantic City, N. J., June, 1925.

The total weight of this atmospheric envelope has been reckoned to be five quintillion Kg (5,000, 000, 000, 000, 000, 000), and here at the surface of the globe the average individual, unconscious though he be of it, is subjected to an external pressure of the element amounting to about 15,000 Kg.

More important for the consideration of the subject in hand, let us recall the fact that in every 24 hours of our existence we take 26,000 inhalations of this element, with a sum total of 26,000 gallons.

Since this is six times by weight the food consumed in the same length of time, it throws some light upon the familiar physiologic truth that a man may live a number of days without food, a number of hours without water, but only a few minutes without air.

That climatic conditions so essential to life must exercise a determining influence upon the health is too self-evident to require argument.

Its effect was recognized by the ancients, and it has been borne out by experience and observation of modern times.

Very naturally it is most evident in disease of the respiratory tract where the causative connection is both immediate and direct.

We are here concerned with the question of the common cold. Most of us have a personal conviction born of experience that weather is in some way responsible for the common cold. We are conscious of the influence of certain bad states of the weather upon our respiratory tract, and we frequently are able to connect an acute catarrhal inflammation with exposure to certain inclement weather conditions, of which the chief feature is an unusual state of coldness, dampness, windiness or sudden change.

The element of low temperature so often seems to play a rôle that the disease is called "cold," from its supposed influence. Indeed, systematic observation of the weather with reference to its bearing upon the frequency of colds universally shows an increase in winter months and in cold weather generally.

Schade, in a statistical study of 750,000 soldiers with reference to the effect of cold weather, found that the incidence of cold was directly proportional to the coldness of the weather

and was parallel with the occurrence of cases of freezing and frostbite.

A very painstaking statistical investigation of the influenza epidemic of 1918 was made by a special committee on "Atmosphere and Man," appointed by the National Research Council of the United States. Employing a delicate statistical method known as the "partial correlation coefficient," designed for picking out and isolating any one of a number of factors in their possible relationship to the epidemic, one of the most definite conclusions reached in the investigation was to the effect that of all factors considered, the state of the weather was the only one which shows any convincing causal relationship to the destructiveness of the epidemic.

We could adduce further evidence of atmospheric influences in the causation of colds, in the recital of many animal experiments, but we do not need further to insist upon the fact that colds are due to weather conditions. Let it be rather to our aim to show, if possible, in what manner these influences operate, for knowledge of this kind might be of great value, by enabling us to avoid certain of these unfavorable conditions or to guard ourselves against those which cannot be avoided.

THE TWOFOLD NATURE OF RESPIRATORY FUNCTION.

The formation of the respiratory organs is Nature's answer to the eternal impact of the atmospheric element. The lowest organism breathes with its whole body, but in the higher species, with specialization of functions, we find an organ especially constructed for respiratory functions.

It is axiomatic that any organ adapted to a special agent, as it becomes sensitive to its influence, must be affected by alterations from its normal manner of acting.

If the eye suffers by reason of bad light, the ear from exposure to intensely shrill sounds, the gastrointestinal tract from indigestible aliments, so we may expect the respiratory tract to be disordered by abnormal states of the atmosphere.

Now let me call your attention to a fact which we believe has a certain significance in connection with our subject, namely, that in the warm blooded animals the respiratory organs have a twofold function to perform with regard to the inspired air.

The distal extremity of this elongated tube is concerned primarily with the chemical constituents of the air; the proximal end primarily with its physical attributes. So that we may, so to speak, look upon the air cells where the interchange of gases take place between the blood and the air, as the chemical laboratory, whereas the physical laboratory is represented by the erectile tissue, for here the physical properties are altered by the addition of the necessary warmth and humidity.

NOSE ADAPTED TO PHYSICAL CHANGES IN AIR. COLD CAUSED BY THEM.

According to all observations, orthodox and secular, it is to peculiarities in the physical properties of the atmosphere that we must attribute the miserable accident we call "catching cold."

We do not hear of colds being caught because of too much carbonic acid or nitrogen or too little oxygen, but because air was bitter cold, or damp and penetrating, or muggy and oppressive, or there was a sharp, chilling wind or an insidious draft; always some extreme or unusual state or some sudden variation in the temperature and moisture. Now, since it is the business of the nose, in performing its function as a respiratory organ, to accommodate itself to the various states of the atmosphere, with respect to temperature and humidity, we might well expect that it would be affected by alterations in these physical states; and since these are the kinds of alterations which seem to give rise to colds, ought we to be surprised that the most common form of cold is the head cold or ordinary coryza?

Air, although a mixture and not a compound, is remarkable because of its constancy of its constituents. With regard, however, to its physical properties, it is distinguished for its variability. It changes not only from season to season, but from day to day, and because of our artificial methods of indoor heating we may be exposed to very sudden extreme changes.

A great burden is placed upon the nasal organ to accommodate itself to sudden changes, not only in the temperature of the air, but in the humidity. Disturbed function must some-

times ensue, giving rise to congestion and eventually to inflammation.

THE NOSE AS AN ORGAN OF HEAT REGULATION.

The nose performs its function of warming and moistening the inspired air in its capacity as a respiratory organ. Secondarily, it has functions of some importance as a heat regulating organ.

Heat regulation is a matter of heat elimination, and since almost 20 per cent of the heat is eliminated by way of the respiratory tract and this chiefly because of the erectile tissue in the nose, we must rank the nose as having a not inconsiderable rôle to play in the regulation of the body heat. In the performance of this part of its function the nasal organ is brought into a very intimate relationship with the skin, which is, of course, the great heat regulating organ of the body.

When the outside air is cold, there is a contraction of the cutaneous vessels, with lessened activity of the sweat glands and consequent diminished escape of heat by way of the skin.

Now, it is just when the outside air is cold that nasal functions are augmented, because the colder the air, the greater the amount of heat it must add to the air inspired.

So a slowing up of the heat regulating function of the skin is a signal for the acceleration of this function on the part of the nasal organs.

This nice cooperation presupposes an intimate connection by way of the autonomic nervous system and indicates to us how the nose can be influenced by atmospheric impressions upon the skin.

Under what we might call normal conditions of the weather —that is, weather that represents no extreme or unusual relationships in temperature and humidity, we have good teamwork between the functions of the skin and the respiratory organs.

The stimulating effect of cold upon the skin should be associated with a harmonious physiologic reaction on the part of the nasal organs.

DISPROPORTIONS IN THE PHYSICAL PROPERTIES OF AIR.

Cold air being naturally drier, there is an equal demand for humidification and calorification, and both demands are met by

the increased activity in the nasal circulation. Let us suppose, however, the cold air to which one is exposed to be at the same time excessively humid. Under such circumstances, it would seem that the respiratory function of the nose would come into conflict with this function as an adjunct to the heat regulating function of the skin. As a matter of experience cold, very humid air is regarded as a very unfavorable meteorologic condition from the standpoint of cold catching.

The excessively dry warm air has likewise a bad reputation, which is justified on the same principle. In this case, lack of stimulation coming from the skin agrees with the lessened need of warming the inspired air, but not with the need for humidification. Consequently, the membranes become dry and disordered.

Warm, moist air represents a proper relationship in physical properties, but it cannot, for other reasons, be considered beneficial.

The nasal organs become sluggish for lack of necessary activity, and the general health suffers because the humidity of the air interferes with the heat regulation and elimination from the body through the process of evaporation.

Civilization, according to Huntington, has always flourished in those countries where the climate is marked by a certain degree of coldness, rather high humidity and frequent cyclonic storms, and by a certain variability, without extremes in either direction.

Health being reckoned as a most important factor in the promotion of civilization, Huntington undertakes the analysis of some millions of deaths in correlation with weather conditions. He concludes that the most favorable weather for health is that of an outside temperature of 64 F., day and night, and a relative humidity of about 80 per cent, day and night, with a fairly high degree of storms and variability from day to day. Tampa, he says, has such a climate at the end of February, New Orleans in March, Asheville in May, Atlantic City in June, Seattle in August, Nantucket and Boston in September, and Portland, Ore., in October.

In the apparent increase in mortality with lowering of humidity as well as temperature, Huntington thinks he finds sup-

port of his theories of the favorable influence of cold and humidity on civilization. This may or may not be true, and, at any rate, the relation of weather to mortality in general is not the same thing as its relation to occurrence of a certain kind of disease.

We have fortunately some valuable statistical evidence of this kind, which bears out our theoretic deduction in regard to the unfavorable influence of cold with high humidity in the causation of colds.

The Committee on Atmosphere and Man, whose accurate methods of statistical study we have already referred to, made a thorough examination of the influenza epidemic of 1918, to determine the influence of temperature, humidity and moisture upon the disease.

The weather data was tabulated for periods of ten days, beginning 70 days before the onset of the disease and continuing 50 days thereafter.

In general, cool and moderately dry weather was found to be associated with a low mortality, and it was noted that an increase of humidity with lowering of the temperature was generally associated with an increase of mortality.

Attention was called, for example, to the marked lessening of the epidemic at St. Paul, Toledo and Grand Rapids, coincident with cool and fairly dry weather prevailing in these localities.

These conclusions are also borne out by certain investigations of this committee made in conjunction with the New York Life Insurance Company, using the same statistical methods.

It was found that in winter high humidity may be favorable to the contraction of pneumonia, from which people die a week or two later.

THEORIES OF THE ACTION OF COLD.

That common colds are more frequent in cold weather and that the low temperature has in some way a direct causative influence cannot be denied, but it has never been settled how this is brought about.

The older writers, following Hippocrates, held that the fluids of the body were congealed by the direct effect of the cold, and

we cannot regard as otherwise than an essential reversion to these ideas the theory of certain modern writers who speak of the effect of cold being due to a colloid injury of tissues (gelosis, frigorosis).

The more generally accepted doctrine at the present time is embodied in the reflex theory of cold; the idea that colds are brought about through the sympathetic, the action of cold upon the extremities causing a determination of blood toward the internal organs, especially the respiratory tract. This explains how the cold might occur at a point remote from the point of action. But the simple determination of blood to a part is not sufficient, even though we admit the second factor, the settlement of microorganisms upon the hyperemic areas. For we cannot look upon cold under ordinary condition as a harmful factor; rather to the contrary it is to be valued for its stimulating effect upon the arterial system and the body metabolism, and its general tonic and invigorating influences.

In order to understand how it may have an unfavorable influence, leading eventually to inflammation, we must think of it as acting in such a way or under such conditions as to cause a disturbance of function.

Determination of blood to a part in accordance with physiologic need gives rise to a normal hyperemia. In physiologic hyperemia there is quickened circulation with contraction of the vessels and heightened function. In congestion, such as may occur from disturbed function, there is a slowing of the circulation, the vessels become dilated and relaxed, which, if it continues, permits escape of serum and leucocytes into the surrounding tissues; in other words, the beginning of inflammation.

We can think of two ways in which mere cold might so upset function as to be a factor for harm rather than good: (1) if so excessive that it paralyzes the vasomotor mechanism, (2) by moderate but long continued action, giving no opportunity for reaction. But it is our contention that colds arise, not so often from the mere effect of cold, as from the existence of unusual states of the atmosphere marked by abnormal relations between the temperature and humidity. Under such circumstances the disturbance is brought about by a conflict of the

nose as a respiratory organ with its function as a heat regulating organ acting in conjunction with the skin.

THE DRAFT.

In the light of these facts, what shall we say about drafts and how explain their supposed influence in precipitating a cold? Is there a real rational basis for the widespread fear of a draft, or shall we regard the popular dread of it as a ridiculous obsession which should not be entertained? Should we still be guided by the old Italian proverb, which says the "air at the window is like a shot from a crossbow" (aria di finestra colpo di balestra); or shall we subscribe without further argument to the pronouncement of certain persons speaking with an air of authority to the effect that draft is nothing more than a dose of concentrated fresh air and it should be, therefore, always welcomed and never avoided?

A draft may be defined as a current of air at a lower temperature and greater velocity than the surrounding atmosphere, impinging upon a localized area of the body. It is the general opinion that it is particularly harmful to a person sitting quietly in a warm, stuffy room, or in any place if the body is overheated or especially if in a state of perspiration.

We believe that herein are given conditions which according to our theory of cold catching we might well expect to bring about an attack.

In the first place, the cold air hitting a restricted area of an otherwise warm body, the effect is to interfere with nature's effort to bring about the necessary heat elimination by way of the skin.

In the next place, the cold air acting through the sympathetic, causes a determination of blood to the erectile tissues, which is contrary to physiologic needs, for the individual is at the same time in warm environment, breathing a warm air.

The result will be a congestion and disturbed function, and if the irritation continues, and the supplementary factor of microorganisms is not lacking, there must eventually result a cold.

The avoidance of a draft, we maintain, must not be considered as arising from an ignorant superstition, but rather as a very wise precaution, having a good scientific foundation.

We hope that our efforts to present the cause of the atmospheric factor in the etiology of colds will not be interpreted as a denial of the value of other factors in the origin of this common and troublesome malady.

Innumerable causes are cited and given emphasis by various writers in accordance with their experiences and their individual bias; these influences must be recognized, though for the most part they should be classed as predisposing or indirect causes.

All causes in the last analysis are external, and we hold that for the common cold we must recognize two kinds as arising from without the body, one as necessary and important as the other, and both acting always jointly in the direct causation of the disease, and these are the bacteriologic and meteorologic factors.

To summarize the argument in the latter case:

The atmosphere as a universal inescapable environment has always and still has profound influence on all life.

For the utilization of this element in the life processes, the respiratory organs were developed. For the warm blooded animals there is a differentiation of function in these organs, in that the distal extremity (air cells) are concerned with the chemical; the proximal (erectile tissue) with the physical properties of the air.

An organ becomes sensitive to the forces of the environment to which it is adapted, and in consequence is affected by alterations therein.

The respiratory tract must therefore be affected by alterations in the atmospheric environment; and the nasal end of it, especially, affected by alterations in its physical properties.

It is exactly the physical properties of the air which are most variable, and therefore the nose has a great burden to adjust itself to these sudden and wide variations.

That the nose, the most frequent site of colds, should be especially affected by changes of temperature and moisture, coincides with the generally accepted view that these are the kind of changes in the atmosphere which especially produce colds.

The nose is concerned with these physical properties, not only as an organ of respiration but as an organ of heat regu-

lation in conjunction with the skin. On account of the latter function it is brought into close relationship with the skin, through sympathetic connection, and this explains how it is readily affected by atmospheric impressions on the body surface.

Under ordinary conditions the nasal organs can adjust themselves to the variations in the physical properties of the atmosphere and perform its respiratory functions as a heat regulating organ, but it must be adversely affected and suffer in the event of unusual and unnatural states, as extremes of heat and moisture, violent variation, and especially in unnatural relation between temperature and humidity.

Prolonged low temperature, for example, is harmful, because it gives no opportunity for wholesome reaction; excessive cold, because it paralyzes the mechanism of reaction; low temperature with very high humidity, because the determination of blood is not then altogether consistent with physiologic function; the draft, because unnatural conditions then exist, which hamper the skin and the nasal organs in the performance of their functions, and disturb the harmonious relation which should exist between them.

We place ourselves then definitely and without reservation in the ranks of the ignorant and unprogressive, who assume that bad weather has somewhat to do with a bad cold; that a cold is, in a sense, properly named, in that low temperature is a big factor; and that one of the surest ways to catch a cold is to expose yourself, while overheated, to a draft.

But further than this I will not go, because I will yield to no one in the advocacy of living an outside life in the fresh open air; in recognizing the tonic invigorating effect of cold air as well as cold water; of accustoming oneself even to the inclemencies of the weather, but always within reason, without excess and avoiding extremes. We do not and cannot live completely in a state of Nature, for our artificial environment has rendered us in a measure unfit for our natural environment.

We have replaced by clothes and warm habitation Nature's own method of adaptability to the air about us, thereby lessening the body resistance and making us more susceptible.

Air indeed, under normal conditions, means much for the promotion of good health, but unnatural states of the atmosphere are potential for harm to man living an unnatural life.

To revert, then, in closing, to the quotation with which we began the discussion: While recognizing that air is, in fact, "the material of all that has had existence," we hold, nevertheless, that the "breeze which gently moves the leaves" may rudely shake the foundations of life; and "the wind which moans through the high forest" moans, perhaps, because it may mean misery to multitudes of men.

XVI.

THE BRONCHOSCOPE AS AN INVALUABLE AID FOR THE PERFORMANCE OF TRACHEOTOMY IN A CERTAIN CLASS OF CASES.*

By Fielding O. Lewis, M. D.,

Philadelphia.

The value of the bronchoscope as an aid in the performance of tracheotomy on a certain class of patients is well illustrated by the following case:

A five-months-old boy was admitted to the Jefferson Hospital on March 4, 1924, with alarming signs and symptoms of obstructive laryngeal dyspnea. The history revealed that the child had suffered with swollen glands on the left side of the neck for several weeks. External excision for the evacuation of pus had been performed on two occasions before being admitted to the hospital. Being unable to nurse, he had been fed small quantities of milk by means of a spoon for the past few days; breathing was labored and noisy. He was restless and could sleep only for a few minutes at a time.

Examination on admission: The child was cyanotic. There was marked respiratory dyspnea. He was septic and exhausted from many hours' fighting for air. There was a large suppurating area in the left side of the neck from recent incisions. A large and rather firm swelling was seen in the region of the thyroid gland. The tissues of the neck were swollen and edematous. The temperature was 102 rectal and the pulse was rapid. The tonsils were enlarged, but there was no evidence of swelling or edema in the oropharynx.

It was quite evident that the child's condition was grave and that immediate tracheotomy was clearly indicated. To operate, however, on the patient of five months with the existing local manifestations, the tissues apparently filled with pus, all landmarks obliterated, seemed to me a hazardous pro-

*Read before the meeting of the American Bronchoscopic Society at Atlantic City, N. J., May 21, 1925.

cedure. It was, therefore, decided to attempt the passage of a bronchoscope in order to inspect the interior of the larynx and to temporarily relieve the laryngeal stenosis. Accordingly, Dr. Clerf, using a four-inch tube, found the epiglottis, left ary-epiglottic fold and the left arytenoid were swollen as a result of inflammation and edema, so that the cords and glottic chink could not be seen by direct laryngoscope. The bronchoscope was then passed with some difficulty through this swollen area. The vocal cords were slightly inflamed, the tracheal lumen was swollen and compressed from the left side. A large quantity of thick secretion was found in the trachea and removed by means of the aspirating tube. The breathing at once improved and the cyanosis became less.

With the bronchoscope in position, the otherwise uncertain and dangerous emergency operation became an orderly procedure. Plenty of time was utilized in the evacuation of the pus before the opening was made in the trachea. The trachea was easily visible, the light from the bronchoscope showing the tracheal wings clearly, which made it possible to do a low tracheotomy. The child, after three stormy weeks, with careful nursing and two life saving bronchoscopies for the removal of inspissated mucus at the cornua, has made a complete recovery.

As I look back over some cases of carcinoma of the larynx, with metastasized glands of the neck in which the trachea was pushed from its normal position and involving the thyroid gland, I can see how much easier a bronchoscope would have aided the very difficult and uncertain operation. This has been done by members of Dr. Jackson's staff with comparative ease and safety. Other conditions, such as an enlarged thymus or extremely large thyroids, with symptoms of laryngeal dyspnea due to pressure, would be likely indications.

XVII.

GLOBUS HYSTERICUS FREQUENTLY A SEXUAL NEUROSIS.

By J. B. Potts, M. D.,

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I wish to present certain evidence which seems to establish globus in many cases as a reflex of sexual origin, due to the use and abuse of the sexual organs, associated with a mental conflict on account of selfishness or of a violation of the laws of morality, society or religion.

(a) I first became interested in this subject during the winter of 1916 when, in conference with Dr. Le Roy Crummer, he suggested that globus was probably a sexual neurosis. Since that time I tried to have a general and neurologic examination made of each case of globus. The result has been that in a large percentage of cases I have been able to establish some sexual neurosis as the basis of the mental conflict which stimulated the nerves of the particular area of the esophagus involved in the syndrome. Furthermore, relief of the symptoms was obtained in those cases in which the mental conflict was eliminated.

Gould's Practitioner's Medical Dictionary defines globus hystericus as "The lump or choking sensation occurring in hysteria caused probably by the spasmodic contraction of the esophageal and pharyngeal muscles." This is, I think, too narrow a definition and should be enlarged to include any feeling of lump, constriction or pressure and at times pain in the throat or esophagus. The fixed ideas of the presence of a foreign body belong in the same group but are usually ushered in by some definite local irritation, as the swallowing of an unusual substance which pricks or scratches the mucous membrane, or an unusually large bolus which stretches the esopha-

gus, leaving an uncomfortable sensation. The severity of the condition varies from the mildest sense of pressure to a spasmodic contraction of the muscles of the esophagus, completely obstructing it temporarily. These severe forms of spasms can be demonstrated by watching through a fluoroscope while the patient drinks a glass of barium solution.

There are many cases of a transient globus. Probably the most common are fear and grief. How many of us have not felt the lump and gripping pain in our throats at the loss of some loved relative or friend. These are normal reactions and quickly pass away.

(b) The symptom complex of globus has no doubt been recognized and described in medical literature from an early date. I hope to search this early literature more carefully, but so far I have been unable to find any mention made of causes other than those given in the following abstracts:

Dr. W. Hamburger, "Stenosis spastica migrans oder intermittens." Klinik der Oesphaguskrankheiten, Erlangen, 1871, p. 94-102:

"There are three grades of stenosis, in the first of which we may place globus hystericus. The patient complains of feeling a spherical body rapidly rising from the pit of the stomach behind the sternum, and then becoming caught in the throat, causing a sort of choking sensation. This spasm is not only painless but entails scarcely any difficulty in swallowing, so that solids as well as liquids easily pass down, although the choking sensation is still present. This sensation usually disappears within a short time, with or without eructation, and reappears at shorter intervals.

"Spasm of the esophagus is only rarely idiopathic; in most cases it is caused by abnormal conditions outside of the organ. Women and nervous men are especially predisposed to it, as well as anemic and chlorotic individuals, and those who are out of condition through age or illness. As a matter of fact, women are much more frequently subject to this spasm than are men. Pregnancy often causes it, but more frequently the cause may be found in abnormal conditions of the genitals, leucorrhea, ulceration at the cervix, oblique position, bending of the uterus, and fibroids of the same; often worms in the intestinal canal, and chronic catarrh of the same are also causes. Esopha-

gismus is also connected with neurosis, and most frequently is an accompanying sign of hysteria, of chorea, of tetanus, of hydrophobia, and of epilepsy (which it precedes, but on rare occasions it terminates the attack). Mental emotion usually brings on the spasm with regard to habitual sufferers; sometimes only the sight of disagreeable food or drinks will cause an attack.

If one examines a woman at the time she complains of feeling the globus, one distinctly hears a sudden contraction of the esophagus and the ascent of a bubble of air.

There can, therefore, be no doubt that the globus is in the esophagus. Because of the weakness of the contraction, however, swallowing is easy."

"Die Tenesme Pharyngien" (A Pharyngeal Tenesmus). Dr. Lennox Brown of London. Archives de Laryngologie et de Rhinologie, 4:8-14, Paris, 1891:

"I doubt whether other authors have employed the term 'tenesmus' to designate a symptom which patients afflicted with certain lesions of the throat often complain of, and which manifests itself by a continual tendency to eject, either by coughing or by clearing the throat, a real or imaginary substance from the pharyngeal part of the alimentary canal."

This is usually accomplished with effort and accompanied by discomfort or real pain. Mucus is expelled, which, sometimes, especially upon awakening, is tinged with blood.

The author has elsewhere shown that this necessity of expelling a substance with difficulty from the pharynx or upper portion of the esophagus in many ways resembles a well known analogous symptom of the lower part of the intestine—i. e., tenesmus. For this reason he has employed the same term.

He has pointed out that what is known as the bulbus hystericus is frequently accompanied by a more or less constant increase in volume of the thyroid body of different degrees, and he has drawn attention to the existence of varices and to hypertrophy of the papillæ at the base of the tongue, or of the lingual tonsil. The sensation of a foreign body or a hysterical bulbus is frequently accompanied by a lesion of the lingual tonsil and of the varices of the same region.

The local causes of the pharyngeal tenesmus may be divided into three groups:

(a) Cases in which a foreign body is caught in some part of the throat.

(b) Cases where there is an evident hypertrophic lesion.

(c) Cases where there is no digestive lesion and which are essentially nervous cases. It is probable that the nervous factor plays an adjuvant rôle in all cases of pharyngeal tenesmus, but it is essential in the third group.

"Schlundkrampf" (Pharyngeal Spasm). Prof. Dr. Otto Körner, Handbuch der Laryngologie und Rhinologie. Publ. by Alfred Holder, Vol. II, p. 335-338, Vienna, 1899:

The globus hystericus appears only occasionally and is not fixed—it rises from the lower portion of the pharynx, at times even from the vicinity of the stomach, and travels upwards and, now and then, downwards. It is spontaneous sometimes, and at others it is the forerunner of common attacks of spasm; then again it accompanies every psychic excitation or appears reflexively while eating. It is undoubtedly produced by an ascending and descending contraction of the esophagus and of the pharynx, and can become a tonic pharyngeal spasm. It is not rare, but, on the contrary, is lacking in very few hysterical cases.

Diseases of the Throat and Nose, Morell Mackenzie. P. Blakiston Sons & Co., Phila., 1844, Vol. 2, pp. 211-219. Spasm of the Esophagus:

Spasm of the esophagus may be (1) a mere psychic or hysterical phenomenon, or (2) it may occur in the course of certain nervous disorders, such as chorea, epilepsy, and especially hydrophobia, or (3) it may be due to some reflex irritation, the cause of which may be either in the gullet itself or at a distance from that part, or (4) it may result from the strain of violent retching.

In cases of spasm, the sufferer sometimes experiences the feeling of a "ball rising in the throat" (globus hystericus), and as long as this sensation lasts nothing can pass down the throat, according to the testimony of a patient. The same patient also called the sensation "hysteria," and in such cases moral as well as hygienic and medicinal agencies should be applied. His mind should be kept employed by regular and interesting occupation, or by change of scene and travel. By

passing a bougie and assuring the patient that there is no obstruction, he sometimes will realize the groundlessness of the sensation. If the disease is believed to be of reflex origin, the cause must be sought out and, if possible, removed.

In several cases of spasm, bromide of potassium has been used with great benefit, but valerianate of zinc in combination with asafetida has proved even more effectual. Diet is of the greatest importance. If the spasm is very severe, thickened liquids should be given, and warm drinks are less apt to bring on the spasm than cold ones; sweetened drinks are more easily borne than unsweetened.

"Hysterische Krampfformen" (Hysterical Forms of Spasm). Dr. Moritz Rosenthal. "Handbuch der Diagnostik and Therapie der Nervenkrankheiten," Erlangen, 1870, p. 245:

The globus hystericus is nothing but a sensory sign of irritation and not a spasm, since the patients are easily able to swallow. In certain forms of pharyngeal spasm it appears because of the constriction of the lower muscular part of the esophagus, perhaps, too, of the circular fibers of the cardia.

Handbook of Diseases of the Nervous System. James Ross. Lea Bros. & Co., Philadelphia, 1885. Page 133, Visceral Hyperalgesia and Paralgesia:

"Globus is a sensation which gives the feeling of a ball ascending from the epigastric region to the throat. It is a frequent symptom of hysteria, and occasionally forms the aura of an epileptic attack. Globus is supposed by some to be caused by spasm of the esophagus and pharynx, and it has consequently been called esophagismus, but the explanation is not a satisfactory one."

Twentieth Century Practice of Medicine. Edited by Thomas L. Stedman. Wm. Wood & Co., New York. Vol. 8 (Article by R. H. Fitz, "Diseases of the Esophagus"). Page 110, Spasm of the Esophagus:

"Spasm or cramp of the esophagus, esophagism, is a localized contraction of the circular muscular fibers, which oftenest occurs near the upper or lower end of the gullet. Closely allied to esophageal spasm and sometimes associated with it is the globus hystericus, which, however, is especially limited to the pharynx."

Differential Diagnosis of Traumatisms and Diseased Conditions of the Esophagus. Thomas Hubbard and E. G. Galbraith, Ohio State Med. Jour., 17:167-169, March, 1921.

To quote from this article:

"The esophagus exhibits freaks of functional perversity, probably due to the fact that it is the connecting link between the sensitive pharynx and a primary organ of the vegetative system, the stomach. The former responds to nerve impulses, volitional and reflex, and the latter is dominated by the vagus and sympathetic systems. It is between the two systems. Reversal of peristalsis, as in emesis, is the extreme physiologic act, and, as a matter of fact, some of the queer perversions of esophageal function are suggestive of abortive attempts at emesis, partial reversal of peristalsis. The mere matter of reaction of contents has apparently a considerable effect in disturbing its functions from the normal. The contents are normally alkalin, and gastric acidity in the esophagus is a cause of persistent globus hystericus. Once this threatens, the psychic factor, excited by the lump in the throat, still further accentuates the perturbation, and we have a rather formidable disturbance. The mere craving for food, of which this is one manifestation, serves to aggravate and prolong the esophagismus. The diagnosis of globus hystericus can usually be determined by study along the lines suggested by etiologic sequence.

"The history of the characteristic stigmata of a psychoneurosis usually clears up the diagnosis. In cases of this character one must take into consideration a trauma or what might be called a near trauma, such as overstretching or that caused by swallowing a very hot or very cold bolus of food. A more or less persistent sensation of lump in the throat may follow such an indiscretion, and in the psychoneurotic this suggestion is overworked."

Diagnosis of Esophageal Diseases. Jacob Gutman. Interstate Med. Journal., 21:1003-1018, St. Louis, Sept., 1914:

One of the reasons for the infrequency of esophageal diseases may lie in the anatomic construction of the organ. Its heavy layer of squamous epithelium, its poor blood and nerve supply, the scarcity of lymph follicles and mucous glands—all favor the organ greatly against ravages of disease. Besides,

the number of esophageal cases is apparently still further reduced because they are frequently overlooked. Quite a number of early lesions are missed because the subjective symptoms they occasion are often mild and, therefore, disregarded. This is particularly true in the case of young women, in whom such complaints as difficulty in swallowing or even discomfort or pressure behind the sternum are wrongly diagnosed as globus hystericus. And yet, the above mentioned complaints are not infrequently the early manifestations of serious disease, such as ulcer, stenosis or even carcinoma.

An Introduction to the Study of the Diseases of the Nervous System. Thomas Grainger Stewart. Bell & Bradfute, Edinburgh, 1884. Page 62, Sensory Impressions:

The writer refers to "various peculiar visceral sensations, which are often of a vague and undefined character, such as a feeling as if the abdominal cavity were a vacuum, a feeling of sinking, a feeling of internal movement, a feeling of universal pulsation, the feelings which lead to a craving for food or for particular and often unsuitable articles of diet. Along with these, the writer draws attention to a common subjective symptom known as globus hystericus—a condition in which hyperesthesia of the nerves of the throat induces a feeling as if it were distended by a globular mass. It is very common during the hysterical paroxysm." Stewart has heard a similar condition described by men suffering from profound emotional excitement.

Pathology of Emotions. Ch. Féré (Translated by Robert Park), London, 1899. Pages 191 and 192:

"Certain muscles which do not act under the influence of the will submit to the same tension and give place to special sensations. It is thus that, under the influence of violent excitation, which comes at the end of pain, the constrictors of the pharynx contract themselves so as to give the sensation of the globus hystericus, which is only, in fact, a pharyngeal tenesmus; so long as it lasts, deglutition is completely impossible."

Nervous and Mental Diseases. Landon Carter Gray. Lea & Febiger, Phila., 1895. Page 438:

"The typical mental symptoms of hysteria are generally supposed to be an alternation of laughing and crying, with an accompanying globus hystericus—i. e., a feeling as if some-

thing were in the throat; and it is generally supposed that the diagnosis must be based upon them. This is a very serious mistake, for, as a matter of experience, it is only a very small proportion of cases in which these symptoms are observed. In reality, the mental symptoms of hysteria in the slighter degrees are practically undistinguishable from those of caprice or lack of self control, while in the graver forms they equal in severity the mental disturbances of such neuroses as epilepsy or catalepsy, such cerebral alteration as is indicated by the different forms of coma, or such as is present in these forms of insanity to which has been given the name of 'psycho-neuroses'."

Neuroses of the Pharynx. F. E. Hopkins, Laryngoscope, 14:506-511, St. Louis, July, 1904.

Neuroses of the pharynx are considered under the heads of abnormalities of sensation, neuralgia, reflex neuroses, spasmodic disturbances, vascular neuroses. Globus hystericus is considered under the first heading, being a form of paresthesia.

"Paresthesia is also common among the neurotic and hysterical. Patients who dwell with morbid apprehension upon the peculiar sensations in the pharynx sometimes develop insanity later, so that the mental side presented with this condition should not be overlooked. The usual complaint is of a tingling or prickling sensation, or of a foreign body as of a fish bone, a splinter of wood, a thread, a bristle, or of the rising of a lump in the throat (globus hystericus)."

Diseases of the Nervous System. W. R. Gowers, P. Blakiston Son & Co., Phila., 1893, Vol. II:

Globus hystericus is an expression of a disturbance in the respiratory portion of the vagus center. It is often described as a ball rising from the stomach to the throat. In other cases it is mere constriction or swelling referred to the fauces or pharynx. When intense, there is probably actual spasm.

Diseases of the Throat, Nose and Ear. Dan McKenzie, William Heinemann. London, 1920. P. 77. Neuroses of the Pharynx:

Hyperesthesia and paresthesia of the pharynx are very common. The patients are generally sufferers from hysteria or neurasthenia. The globus hystericus is the most typical hysterical paresthetic condition, but all kinds of feeling may be

referred to the throat by hysterical or nervous people. Every care must be taken to exclude organic disease in the supposed functional neuroses of the pharynx. Incipient phthisis or malignant disease may be responsible for the perverted sensation, and all patients with such a symptom must be examined so as to exclude these diseases. It is a wise rule always to make an esophagoscopic examination in women with paresthesia pharyngis. In a certain number of cases the sensation seems to be due to enlargement of the lingual tonsil. It is not infrequently removed or relieved by the application of the cautery to this area.

For the paresthesia of nervous people, general treatment alone should be adopted. Operative procedures on the pharynx are harmful, as a rule, except in the reflex conditions which are clearly due to hypertrophy of the lingual tonsil, in which case that disease should be treated.

If a patient is convinced of the presence of a fish bone or other foreign body in the pharynx, it is legitimate, sometimes, to provide such a body for his satisfaction. Cure may follow. If, after thorough examination, repeatedly carried out, no organic basis is found for the sensation, the patient should be told that his disease is neither cancer nor tuberculosis, as the fear of these diseases is often responsible for the paresthesia.

Modern Medicine. Osler and McCrae. Lee & Febiger, 1914. P. 87. Neuroses of the Esophagus:

Etiology.—Spasm which is purely neurotic, called idiopathic, exists chiefly in young adults, rarely in children, and oftenest in females. Excitable, nervous people and hysterical persons are the ones most frequently afflicted, and shock, fright or anger is often the signal for an attack; the older writers seldom fail to mention the dread of hydrophobia as an excitant. Pregnancy appears at times to have this disturbing effect. There are also cases which are not idiopathic but reflex, occurring generally in the same kind of people as the idiopathic. As a result of errant stimuli, excited by a diseased condition of structures which have a related nerve supply, or stimuli arising in a functionally disordered central nervous system, or even stimuli originated by some slight lesion of the esophagus itself, the motor nerves of the esophagus carry impulses which result in a spasmodic contraction of the muscles, occurring most often

at the upper part of the tube or at the cardiac orifice of the stomach. Notwithstanding the reality of the muscular contraction, the cases which have died with a spasmodic stenosis have failed to show any sign at autopsy, although one or two reported cases have indicated a possible persistence of the condition after death. The most usual site of spasmodic closure of the esophagus is at the cardia, and whether this be considered esophageal or gastric matters not for practical purposes. These stenoses are found in various parts of the tube, and the spasm is not necessarily always at the same place. When an organic stenosis exists, a spasmodic stenosis at the same or different part of the tube may be superadded.

Diseases of the Nose, Throat and Ear. Ballenger. Lea & Febiger, 4th Edition, Chapter XIX. Page 364-365, Neuroses of Sessation:

Tonsillar disease is often the cause of it, hence these organs should be thoroughly examined for diseased conditions. The passage of a bolus of food or foreign body may cause an abrasion, which may be followed by the sense of a foreign body in the throat. The menopause is frequently attended by perverted sensations in the pharynx. Patients at this period sometimes complain of the sensation of a rope or hairs in the throat. Hyperplasia of the lingual tonsil seems in some cases to cause it. The same is true of elongation of the uvula.

The perverted sensations complained of are cold, heat, a foreign body, itching, tickling and the dislocation of the essential parts of the fauces and pharynx. Patients sometimes complain of swallowing the soft palate, etc. Most of the female cases seen by me have suffered from melancholia during the menopause and have had a suicidal tendency. One patient committed suicide by drowning, some months after she passed from under my observation. The paresthesia may be so marked as to cause a distressing cough and laryngeal or esophageal spasm.

The abstracts quoted cover the subject as given in the literature, in a fairly comprehensive manner. It is evident that the syndrome known as globus hystericus has long been recognized as neurosis, but apparently has never been definitely fixed as a reflex due to a mental conflict. It is my firm con-

viction, after watching a large number of patients, that the syndrome we recognize as globus hystericus is always a reflex secondary to a mental conflict. This conflict is due to some act, circumstance or condition which is in conflict with that individual's self respect or moral or religious training. The thing is of such a nature as to cause the individual to wish to suppress it. The trauma to the mind is the essential factor. Local conditions of the throat are incidental and not etiologic factors.

Obstruction due to foreign body, paralysis, tumors, ulcers and injuries, all are pathologic conditions and must not be confused with globus hystericus.

Every normal person has experienced the sensations of emotional projection to the very zone in which victims of globus localize their disturbing sensation. With a little self analysis each one of us may easily remember the definite projection area of any or all of the most diversified but definite emotions.

The biblical expression, "The nobles held their peace and their tongue cleaved to the roof of their mouth" (Book of Job, chapter XXIX, verse 10), well recognized as a description of extreme fear, is cited as an example of the wonderful psychology of the old testament. The "sinking sensation in the pit of the stomach," caused by exposure to temporary or transient danger, shows that there is along the entire topography of the esophagus an area, the site, not of individualistic, but of unusual seats of emotional projection: anxiety, fear, rage, sadness, lonesomeness, homesickness, guilt, etc. Each and all have their exact spot of emotional projection recognized as clearly by the individual as the various forms of facial expressions are recognized by the audience at the "close up" of the movies. While these are common experiences of mankind, the individual presenting himself in our office with globus has suffered the same temporary shock or insult to which we are all subject, but the persistence of the manifestations after the normal period is passed is the important consideration for our investigation. The general rule, that the hysterical manifestation persists only when the distress, however great it may be, is less than the mental distress caused by the primary thought, must always be borne in mind in analyzing these cases, since, in this rule and this rule alone, can we find a reason for the persistence of the symptoms called globus.

Whatever the disturbing factor may be, it serves in the main only to determine the localization of the globus, but in the localization the keen physician gets his first intimation as to the nature of the mental conflict predisposing to such hysterical manifestations.

In the great scheme of the universe, in order that the species may survive, reproduction and the desire to live are the strongest instincts of the animal world. Hence the desire to copulate, that reproduction may take place, is the strongest inherent instincts of the human race. This being so, it naturally follows that any act of suppression or perversion of the normal functions, or insult to the organs, will react in a most profound manner upon the reflex nervous system.

The vagus nerve is the nerve of emotion and responds in an unusual degree to these stimuli, as evidence, the rapid or pounding heart beat, increased respiration under excitement of emotion, the lump in the throat, or gripping pain in the larynx, or sickening weight in the socalled pit of the stomach, inspired by fear. That the esophagus is most frequently affected is probably due to its function, plus its musculature and innervation.

In recent cases the localization hint, coupled with some general knowledge of human nature, is usually sufficient to enable the physician to relieve the case by well considered advice and proper explanation. When the case, however, has persisted for any length of time the primary mental conflict is so buried under a mass of added and artificial conflicts that it may be extremely difficult for even the most experienced psychologist to determine the exact nature of the primary conflict. It may be assumed, and experience bears out the assumption, that this primary conflict is almost always in the sexual *ideation* of the individual. Even if sexual trauma be established from the anamnesis, this is not always a sufficient explanation for the particular reaction of globus determining the particular case. The individual's *moral* reaction to his individual trauma must always be investigated. When no history of sexual trauma can be elicited, it is, at the best, only negative evidence, and forces the physician to a deeper analysis of the individual's moral reaction to the sex problem in general.

In the individual reaction to the fundamental sex *drang* every element of training, education and reaction to environment plays its part. None of these is perhaps more important than the religious training of childhood. And the respressive religions furnish a fertile field for the development of psychoasthenic disturbances. In individuals subject to this religious repression a sex thought no greater than that constantly unconsciously handled by individuals with normal training, can easily be sufficient in itself to constitute by its reaction upon this fundamental training a sex trauma; and it is frequently possible to determine the church affiliation of individuals after a cursory recital of their symptoms.

This has a very practical bearing upon the relation of the laryngologist to the patient, and I firmly believe that before any patients, suffering from a globus, are subjected to any form of treatment, especially surgical, they should, after a careful examination, be referred to a competent neurologist for an analysis to determine, if possible, the basic mental conflict which is the real cause of the globus. I have been guilty of removing tonsils, as advised in our textbooks and literature, only to find the trauma to the throat had fastened the reflex more firmly than ever, and that I had the credit, which I really deserved, of making my patient worse. I have examined patients in which the same thing has happened to my confreres. Most everything in our operative armamentarium has been done to these poor sufferers—uvulotomy, submucous resection, drainage of the various sinuses, local applications to the pharynx, diets, tonics, etc. The latter two, at least, did no harm.

I saw one young lady that had been having daily applications of 20 per cent silver nitrate, to the pharynx, for more than a month. For two weeks she had only been able to swallow liquids, and for the past five days had had no fluids by mouth. Her physician then pronounced the obstruction carcinoma or sarcoma, and she came to me, fully expecting to have the diagnosis confirmed, and her mind made up that she must die. No pathology was found and she was referred to Dr. G. A. Young. Five days of psychotherapy restored her normal mental balance, she gained ten pounds in weight and left for her home, relieved of her globus and eating and drinking without apprehension or difficulty.

Another young lady had had a bougie passed down her esophagus daily, for a year, in order that she could eat one meal. Examination revealed no pathology, and a single conference with Dr. G. A. Young relieved the globus so that she could eat. She returned to her home the following day, and has had no recurrence of the trouble in two years.

The following cases are given to illustrate the different causes encountered. All have been carefully examined to eliminate pathologic conditions:

Case No. 1.—E. O., male, 18, high school student, lives on a farm. September 13, 1919. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: For past three months has had a feeling of something in throat. Cannot swallow and has to cough. Says coughs to get it up and swallows to get it down. Very long prepuce. Onanism. Otherwise normal.

Case No. 2.—W. H., male, age 21, waiter, single. August 17, 1923. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: Last February had sore throat for one day. Left lump in middle of neck and soreness in windpipe. Feels heartbeat running up to ears. Scotch-Irish; Catholic; emissions; onanism.

These boys had both been practicing masturbation for some time, but it was not until after they were told of the injury to themselves and the moral wrongdoing that they developed remorse and mental conflict resulting in globus. The condition was relieved in both cases by rational explanation and advice. The haphazard teaching and awful picture painted by the Y. M. C. A. teachers and others who undertake to inform and train our youth without any understanding of the medical science or sex psychology is a most dangerous thing.

Case No. 3.—C. P., age 38, female, white, farmer's widow, German Lutheran. Laryngorhinologic examination negative. Referred to Dr. Crummer. Present complaint: Persistent lump in throat for four years. Never a day without it. Sleeps well; dreams a lot. Four children; youngest, 10 years old. Husband died five years ago. Lump came three weeks after tubal pregnancy, four months after husband died. Free for a year, then came back suddenly while working out. Illicit intercourse with farmhand.

This woman became pregnant after the death of her husband from having intercourse with a farmhand in her employ. Remorse and anxiety resulted in globus, which was relieved when the mental conflict was stopped by an operation to remove the ectopic pregnancy. She was entirely well for a year, and then the lump suddenly returned when she went to work, as a general maid, on another farm and developed a fear of meeting the farmhand who had seduced her. The conflict in her case was emphasized by the strict teachings of her religion.

Case No. 4.—V. B., female, white, age 30. October 10, 1922. Laryngorhinologic examination negative. Referred to Dr. G. A. Young. Present complaint: For a number of years has felt a burning high in throat, back of soft palate. Feeling of pressure in larynx. Difficult to breathe at time. Dr. Young's report: Globus, secondary to being raped several years previously, by her employer, in the back room of his store. Since that time has never had any gentleman company or gone out in a social way. General appearance very bright, vivacious and exceptionally good looking.

Case No. 5.—H. G., male, white, age 40. October 3, 1923. Present complaint: Since last February has had trouble in swallowing; more difficulty with liquids than solids. Feels as if he was going to choke when liquids or bolus touches soft palate or pharynx. No trouble after it passes this region. Pharynx and larynx normal. Referred to Dr. G. A. Young. Dr. Young's report: Globus. Married previous to entering the army. Divorced wife after returning home on account of some act of hers which he felt was very wrong. Has always been very much attracted by women. Passionate nature. Liberal social views previous to marriage. In France saw the effects of lues and received a great mental shock. During service, and since returning has been afraid to have any sexual intercourse. Very much attracted by women and afraid to be left alone with any one woman. Always insists on being in a crowd.

Case No. 6.—J. L. P., female, white, aged 30. August 15, 1923. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: Illness of about two or three years' duration. Married ten years. Has one child, aged nine. Weight formerly 135 pounds; now 120

pounds. Has gas in stomach. Can't breathe. Has pain in right side and ovaries. Bowels constipated. No mucus seen. Appetite not good. Distress immediately after eating. Lately can hardly swallow on account of lump in throat. Sleeps poorly, restless and dreams a lot. Practices withdrawal. Globus. Has constant fear of conception.

Case No. 7.—A. G. S., female, white, aged 39, housewife. October 11, 1923. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: Lump in throat; worse at night; often keeps her awake; some difficulty in swallowing. Is four months pregnant. Has two children, 15 years and 2½ years of age. Had same condition when pregnant, during the last three months of pregnancy, with last child. Globus. The mental conflict in this case was due to the fact that she did not want any more children after the first one.

Case No. 8.—Miss E. T., female, white, age 29, teacher. August 11, 1917. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: Has had a feeling of lump in throat for six years. Not so bad for past three years. Usually during the night. Last attack at midnight. General health good; physical examination negative. Globus. Cause: Dreams in which phallus symbols occupied a prominent place. Brought up strictly in the Christian Church. The reaction of such dreams on her natural modesty, plus her religious training, produced a mental conflict resulting in globus.

Case No. 9.—Mrs. W. P., white, age 28, housewife. October 17, 1919. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: Lump in throat. Says she swallowed a pill, a year ago, which has stuck in her throat. Located by her just below the suprasternal notch. General condition good; physical examination negative. Globus. The pill which she swallowed was given her to produce a miscarriage. Instead of abortion, she had a hemorrhage from an ectopic gestation, was operated upon and recovered except for the globus.

Case No. 10.—Mrs. J. M. H., white, age 42, housewife. August 10, 1917. Laryngorhinologic examination negative.

Referred to Dr. Le Roy Crummer. Present complaint: Throat aches and pain in ears. Lump in throat comes and goes. Feels scratchy at times. Began two years ago after coming home from summer vacation at a lake. Globus. Cause: Remorse and fear of meeting man she had intimate relations with at lake. April 13, 1918. Globus better.

Case No. 11.—M. B., male, married, age 44. August 11, 1917. Laryngorhinologic examination negative. Referred to Dr. Le Roy Crummer. Present complaint: Lump in throat for two years; worse in morning; can't eat. Globus, due to withdrawal.

Case No. 12.—Mrs. M. A. D., white, age 35, housewife. March 17, 1919. Laryngorhinologic examination negative. Referred to Dr. Crummer. Present compaint: Lump in throat which comes on soon after getting up. Can feel it all the time. Never completely blocks swallowing. Worse since having tonsils removed in April. Married nineteen years; five children; youngest three years old. No sexual intercourse with husband since birth of last child. Husband unable to work on account of inflammatory rheumatism, for past six months. Considerable financial worry. Globus, the result of "repressed desire," to avoid conception, plus worry.

These cases were selected from a series of more than one hundred, to illustrate the most common causes.

Globus following trauma is no different from any other globus. The trauma simply serves to furnish the patient an explanation of his condition, the real cause of which he has tried to bury so deep that it cannot even be suspected, but which still survives to trouble the subconscious, and often, the conscious mind.

SUMMARY.

(a) A true syndrome globus hystericus is a neurosis secondary to mental conflict.

(b) This conflict is usually secondary to some mental trauma received from an act associated with, or a condition of, the reproductive organs.

(c) No surgical procedure, other than passing a bougie, should be resorted to until the true etiology of the condition is ascertained and relieved by a competent neurologist.

Thanks to Dr. Le Roy Crummer and Dr. G. Alexander Young, of Omaha, for their very valuable assistance.

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SOCIETY PROCEEDINGS.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Monday Evening, October 19, 1925.

THE PRESIDENT, DR. HARRY L. POLLOCK, PRESIDING.

DR. ARTHUR J. CRAMP, Director of the Bureau of Investigation, American Medical Association, presented a paper on:

"Aural Quackery and Pseudomedicine."*

DR. HARVEY FLETCHER, of the Research Department, Bell Telephone Laboratories, addressed the Society on

"Audiometry,"†

illustrating his remarks by means of charts.

DISCUSSION.

DR. J. GORDON WILSON: At first glance the two parts of the program appeared to have little connection, but this was not so. Quackery can only thrive in ignorance, and ignorance can best be combated by the results of investigations carried into the realm of the doubtful and unknown. Quackery is not always as crude as Dr. Cramp's paper suggested. It is often subtle and persuasive. To meet quackery one must be well informed and ready to state the truth with simplicity and with patience. But to fight quackery is but a small part of our duties. We must be ready to heed the cry for help to which quackery responds. One loses patience with the superior otologist who complacently tells his patient that for a particular malady nothing can be done—and lets it go at that. The cry for help is often urgent and pathetic, and until we answer it painstakingly, honestly and intelligently, some of the responsibility for quackery rests on our shoulders.

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*See page 91.
†See page 165.

Dr. Wilson confined himself chiefly to Dr. Fletcher's discussion, stating that until recently the fascination of electrophysics overshadowed the realm of acoustics and few physicists devoted much of their time to the investigation of the properties of sound and their application. Now the practical application of electrophysics to acoustic problems has enriched the domain of otology.

It has long been recognized by physicists that the estimation of the minimum audibility of sound at all pitches is of prime importance in the study of audition. But the range of tones to be examined, and the difficulty of obtaining suitable apparatus capable of examining such audibility over so great a range, have proved a great barrier. Not only is the estimation of the minimum audibility of sound at all pitches important for the understanding of audition, but the ascertaining of the minimum audibility at various pitches is of importance in otology, to determine the degree of impairment of hearing, to localize the pathologic changes where clinical examinations may fail and to estimate the results of treatment. Tuning forks have been our mainstay, but tuning forks have great limitations. They are wanting in scientific accuracy since we cannot well measure the amplitude of their vibration. Each vibration period varies in each set and alters with use. It is impossible to give a percentage estimate of the impairment of hearing compared with normal by means of forks, for the amplitude of the tuning fork, when it ceases to be heard in the two ears, is difficult to estimate.

Until recently, we have had no satisfactory means of measuring the intensity of the sound perceived. There was no certainty in regard to the position in the sound scale in which the maximum sensitivity for sound perception lies. By graphs, such as the audiometer gives, we can get such knowledge in normal and defective ears, and an endeavor can be made to apply data so obtained to pathologic conditions in the ear.

What impresses one is the cooperation that now is taking place. The perfecting of the telephone and the problems of wireless communication have compelled the electrical engineer to take careful note of the mechanism of hearing. He seeks to understand the anatomic structure of the hearing organ and its mechanism, and recognizes that such information can

best be obtained from the otologist. This association is compelling the physiologist and otologist to endeavor to grasp the physical problems which the ear presents. A very simple example of the results of such an association and interchange of ideas has been the very plausible suggestion made by an electrical engineer, who has paid very considerable attention to acoustic problems, that in the diaphragm of the loud speaker we have a structure analogous in physical properties to the drum membrane.

From this association, otology is destined to receive great benefit, and we already see an awakening of interest in the fundamental problems of hearing. It has been characteristic of this reawakening that the work in this country has not been concentrated in one area, and that it has not been confined to the pure scientist. The pioneer work of Professor Seashore (to whom we owe the name audiometer) stimulated Dr. Dean and Dr. Bunch in Iowa. The laboratory at Geneva, Illinois, where Dr. Sabine for many years has worked on problems of acoustics, now gives willing aid to the otologist. In Los Angeles, the experiments carried on by Dr. Knudsen and Dr. Jones, not only have given us scientific data of importance, but have furnished practical points of far reaching significance. The electrical engineers of the Bell Telephone Company not only are in constant touch with otologists, but are showing their desire to cooperate with them fully and freely.

Dr. Fletcher had told a little of the thought and patient work that has gone into the building of the Western Electric audiometer. There are many practical points in this instrument which should appeal to the otologist.

1. The instrument offers simplicity in operation.

2. It produces a series of pure tones whose frequency or pitch can be varied in steps throughout the scale of audition (from 32 to 16,000 vibrations per second in 20 logarithmic steps).

3. The loudness of each tone can be varied in steps throughout the tone. The intensity (loudness) as it strikes the drum membrane is measured in sensation units which can readily be transformed into absolute units (dynes). There is no standard sensation unit to measure hearing, and there is diffi-

culty in comparing audiometric measurements from various investigations. Once a standard is accepted the present confusion in records will disappear. In this instrument a logarithmic system of units (loudness) is adopted, which the Western Electric engineers believe will answer well the needs for a practical system.

4. By means of charts supplied, the findings may be quickly plotted, and the amount of hearing loss clearly indicated.

Dr. Wilson thought the steps in frequency in the upper limits might well be smaller. Jumps of two or three thousand vibrations seemed rather great. Breaks have been found occurring within such limits which have a meaning to the otologist. For example, a marked depression in the hearing, a socalled "island of deafness" occurring about 3500, could not be detected by the instrument. The steps for the loudness appeared to be adequate.

He had made a number of audiometric charts, but had found it difficult to synthesise them into definite types corresponding to definite ear lesions. The possibility of a synthesis was to be expected—how completely, he did not know. The following were some of the practical findings made apparent by his audiometric charts:

1. In all forms of deafness the region of hearing of speech (the middle range) is more resistant to disease. This appears to be due to the fact that here lies the minimum intensity to produce the sensation of hearing.

2. The usual teaching that nerve deafness affects the higher tones, and conduction deafness the lower, requires modification. In many cases of conduction deafness the higher notes go early and in nerve deafness the whole scale may be depressed.

3. In otosclerosis some of their charts indicate that one of the earliest signs is a contraction of the upper tone limit. It may be some time after that the lower notes get markedly affected, and that we can be sure we are dealing with that clinical entity called otosclerosis. To him this characteristic has been one of the most interesting findings brought out by the audiometric charts. Further observation is required in regard to its frequency, for it obviously has a bearing on the genesis of this intractible disease.

4. Choked labyrinth has particularly attracted his attention. That it exists he has no doubt, but one hesitates to say that it will yield data comparable to the choked visual disc. In tumors of the posterior cranial fossa hearing is almost universally lowered. He was not prepared to synthesise his findings, but thought the following points in the graphs were of interest. The only case in which they found a complete tone gap occurred in a tumor of the cerebellum. It was present on both sides and extended from 1600 to 2700 d. v. After decompression operation, the gap was narrowed and hearing improved. Another characteristic is the rapid fall of the curve of hearing in frequencies above 3500, and with it the disappearance of hearing of all sounds. A mathematician, without knowledge of their cases, was asked to classify a series of their graphs in which were included cerebellar cases, and he grouped all their brain tumor cases in one group of three. He wished it clearly understood that his observations have been made on too few cases to be anything but suggestive.

5. A scientific basis is here presented on which devices may be built to suit various types of deafness. Though little can be reported as yet, he hoped the time would come when the otologist will write out the audiograms and the electrical engineer will be able to supply the aid best suited to the needs. It is well to recognize that many factors enter into the fitting of hearing devices of an electrical nature, as Dr. Fletcher had stated in a recent paper. In addition to audiometric measurements there has to be ascertained the loudness best suited in a particular case to interpret speech, for great loudness over the telephone introduces subjective tones which interfere with adequate interpretation.

Dr. Wilson believed that at no time in the history of otology has the outlook for progress been so bright. It requires neither a prophet nor the son of a prophet to predict the oncoming of waves of increasing knowledge in the physics of audition and in the pathology of ear disease. To meet this the clinical otologist must be ready to apply the information so obtained to the patients he is called upon to treat.

Dr. C. C. Bunch, Iowa City, Iowa, said that he had been much interested in the problems of auditometry since 1917. When the pitch range audiometer was demonstrated to the

otologic fraternity at Atlantic City in 1919, the reception on the part of many workers, along the clinical lines of functional tests, was rather problematic. The presence of the large group of men and their close attention to the presentation by Dr. Fletcher had shown that medical men are interested in those problems and wish to have the aid of the physicists in the solution of the problems of sound measurement.

But physicists have also shown an awakening in this line of research. Few, however, are actively engaged in the problems of sound if one may judge by the number of articles presented on the subject of sound in the Science Abstracts. The field of general physics and electricity are of much greater interest to them. A few years since, a physicist asked an official of the Western Electric Co., as to the extent of their interest in the problems of acoustics, and he was told that they were not at all interested. But now their interest has been aroused, even to the extent of supporting an immense research laboratory with a great staff of engineers.

Relative to Dr. Fletcher's remarks concerning the function of the ossicles in the hearing process, Dr. Bunch thought it was difficult to estimate this effect on the evidence presented in the audiograms. He stated that he had found cases which, after the radical mastoid operation, showed very little loss of hearing, some were quite hard of hearing for tones throughout the entire range, while others were hard of hearing for high tones. Unless we are able to ascertain the functioning power of all parts of the conductive and perceptive mechanism it is difficult to determine the function of any one part. This is a problem which will require the careful support and study of many workers in the field.

Dr. Max Goldstein, St. Louis, Missouri, said that he had been very reluctant about giving up the tuning forks, the Galton whistles and watch test, but had been intensely interested in the audiometer and had become a complete convert. This not only because it gives everything they had before in the testing media, but because in addition it affords a means of quantitatively measuring sounds. This the audiometer does accurately, and it is the one advantage that has been developed in otology in the whole plan of the functional test of hearing.

A number of practical deductions have been developed from the audiometer.

Briefly, these are a means of differentiation of the types of deafness which are familiar to all in the more definite gross forms, new types of conduction mechanism impairment, and a graph for the low tones. The graph can be verified by tuning forks and whistles. If there is some reduction in the high tone scale, it is evident that one is not dealing with nerve deafness. If there is a reduction of the low pitched sounds and a fairly well marked approximation to the normal in the middle of the scale, and again, a reduction in the high pitched sound, one is dealing with otosclerosis. These three types of graphs may be considered classical and are entitled to all of the consideration that can be given them.

In the children with residual hearing there is in the speech scale a tone that goes from 300 to 3,000 total vibrations per second. If a deaf child still has, even in the lower part, a hearing capacity within these limits, there is still some hope of stimulating the hearing by some mechanism which remains to be developed.

Dr. Goldstein thought the bone conduction apparatus that is being developed in connection with the audiometer would, perhaps, need a different standard, but there is a possibility in the development of the telephone receiver that will be just as helpful as the present tests.

He expressed his homage to the electric engineers of the Western Electric Company, particularly for their very scientific and helpful work in connection with the medical profession. In all cases they have practical evidence that the audiometer and the otogram will be of inestimable value in otology.

Dr. Fletcher (in closing) said that he had talked on the data which might be obtained with an audiometer and not the machine itself. He had tried to point out the new scale for measuring hearing which could be used in the graph form called the audiogram. This can be done with tuning forks as well as with other kinds of audiometers. He stated that the Bureau of Standards was now ready to calibrate forks so that with them one can get directly hearing loss on this new scale. To do this, one only had to take the difference

in hearing times for the normal and for the patient, and multiply by a constant. Probably as accurate results can be obtained with the tuning forks as with the audiometer if one is careful and takes sufficient time, but one can get the same result with the audiometer very much more rapidly. In a good many cases, however, tuning forks will still be the most practical thing to use, but he suggested that the data be expressed in terms of this new scale, then the results of the different observers may be directly compared.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY.

Meeting of Thursday Evening, November 19, 1925.

THE PRESIDENT, DR. HARRY L. POLLOCK, PRESIDING.

The program was devoted to a consideration of the deafened.

Introductory Remarks.

DR. WENDELL C. PHILLIPS, President-elect of the American Medical Association, was introduced by the chairman, and expressed his pleasure at being a guest of the Chicago Laryngological and Otological Society and his interest in the program to be presented. He said that there was a phase of otology that should come very close to the heart of the otologist, but which was shown to a very slight degree, that is, the social service work among people afflicted with deafness. By this he referred to organized effort to bring the deafened people opportunities for education, for proper occupation and for social rehabilitation. He devotes considerable time to work among the deaf through the New York League for the Hard of Hearing. There are hundreds of children in every city with defective hearing, whose hearing can be restored if they are discovered in time. A great deal of satisfaction will come from such work.

He expressed his appreciation of the honor which had been conferred upon him by the medical profession of this country and stated that it was a satisfaction to know that he was the first otolaryngologist to be president of the American Medical Association.

DR. GEORGE E. SHAMBAUGH presented

Statistical Studies of the Children in the Chicago Public Schools for the Deaf.

These studies were undertaken for the purpose of determining what light if any could be thrown, from the study of statistics, upon the many questions relating to the children in our institutions for the deaf, such questions as the occurrence of partial or total deafness, questions of the relations of these to congenital and acquired cases, the relation between those

occurring with otitis media and those which developed quite independent of this process, statistics in the etiology of acquired deafness. Various charts were devised, setting forth in a graphic manner the full relationships which were studied.

A brief summary of the general conclusions are expressed as follows:

Exactly one-half of the children examined were found to be congenitally deaf, while the other half had acquired ear trouble.

2. Total deafness existed in 57.9 per cent of the congenital cases and in 35.2 per cent of the acquired cases.

3. Partial deafness was found in 42.1 per cent of the congenital cases and in 64.8 per cent of the acquired cases.

4. Vestibular responses were present in 75.1 per cent of the congenital cases and in 72.93 per cent of the acquired cases.

5. Vestibular responses were absent in 24.9 per cent of the congenital cases and in 37.93 per cent of the acquired cases.

6. A total absence of vestibular responses and total deafness were found in 17.5 per cent of the congenital cases and in 26.2 per cent of the acquired cases.

7. Presence of vestibular responses and total deafness were found in 40 per cent of the congenital cases and in 8.2 per cent of the acquired cases.

8. Absence of vestibular responses and partial deafness were found in 6.2 per cent of the congenital cases and in 11.03 per cent of the acquired cases.

9. Presence of vestibular responses and partial deafness were found in 34.5 per cent of the congenital cases and in 53.8 per cent of the acquired cases.

Acquired Type of Deafness.

DR. J. W. HOLDERMAN presented five cases illustrating the acquired type of deafness. The deafness in each case was ushered in by a bilateral otitis media.

The first child showed a bilateral defect in hearing, with normal vestibular responses. His history was as follows: At the age of 1½ years, he developed an acute bilateral otitis media without any definite infectious disease accompanying it; this soon subsided, with the continuation of a practically normal drum membrane on each side. The lower part of the

bone range was practically normal; the marked deformity was a lowering of the other tone scale of 9.5 and 10, respectively, with the Galton whistle.

The second child developed measles at the age of three years, followed by an acute otitis media. The defect in hearing appeared soon after, and at the present time was quite similar to that in the first case. However, the whispered voice was not heard except very close to the ear. There was a question whether C_4 was heard in either ear, and the Galton whistle was not heard. The vestibular mechanism revealed a quite marked diminution. There was a marked lowering of the tone limit, which was apparently brought about by a primary neuritis rather than by the otitis media.

The third child developed scarlet fever and diphtheria at the age of five years, and soon after a bilateral otitis media, which necessitated two operations on one side and one on the opposite side. The right ear had discharged since the operation; the left one only at times. Hearing on the left side was practically gone, and there was total defect of the drum membrane. She had a vestige of hearing on the right side. The spoken voice was heard at from 9 to 12 inches; the large C was not heard. C_4 was very much shortened; the Galton whistle was heard at a range of from 4 to 25. There was some question as to the vestibular mechanism; the responses were so negligible that the case was classed as having a total loss of vestibular function.

The fourth child developed pneumonia at the age of 1 year, with bilateral suppurative otitis media, which still persisted. He said a few words at one year. He developed measles at the age of 3 and later had a mastoid operation on the left side. There was unequal nerve deafness and chronic bilateral otitis media. He had total defect on both drum membranes and also total loss of hearing. The perception mechanism was not active, and vestibular mechanism was reduced about half. There was nystagmus of 12 and 14 seconds, respectively.

The fifth child developed scarlet fever at the age of 5, with bilateral otitis media, and later a mastoid operation on the right side. There was complete absence of drum membrane, also total loss of hearing and total loss of vestibular reaction.

Dr. Holderman then showed four children, members of one family, illustrating congenital deafness. These children had never talked and had never been able to hear. It was an interesting example of consanguinity, the parents being first cousins, with no deafness in the family, so far as they were able to ascertain.

Definitely Congenital Deafness.

Dr. Alice K. Hall presented patients with definitely congenital deafness.

GROUP I.

Case 1.—Mary M., age 15, had normal vestibular reactions and bilateral vestiges of hearing, the greater in the right ear. This was the most common type of congenital deafness found in this group.

Case 2.—Sam D., age 17, had normal vestibular reactions. The after-turning nystagmus was 30 to 35 seconds, but he had a total absence of hearing.

Case 3.—Anna K. had an almost total loss of both vestibular responses and hearing. She did not respond to the turning tests performed in the usual manner, but on increasing the speed and number of vibrations and with the head 120 degrees forward there was a nystagmus of about 5 seconds duration. She gave no response to the functional tests for hearing with voice, tuning forks and Galton whistle, but a 3 per cent hearing was revealed in the right ear by the use of the 2A audiometer.

Case 4.—Wittold G. represented the most uncommon type of congenital deafness, namely, a total loss or absence of vestibular responses, with the presence of a vestige of hearing, in the right ear only.

Three of this group come from families of deaf children. Case 1 was especially interesting, for the child came from a family of seven children, five of whom were born deaf, while two had normal hearing. The parents had normal hearing. Two of the deaf children died in early childhood. Three living deaf children, all girls, were in attendance at Beidler School. The one shown was the eldest and had the best hearing and greatest vestibular responses. The second girl, 10 years old, had a greater degree of deafness and loss

of vestibular responses, while the baby, 5 years old, was apparently totally deaf, with an after-turning nystagmus of only five seconds.

Consanguinity was not a factor in any of these cases. The blood Wassermann of each was negative, and there were no clinical signs of hereditary syphilis. So far as the parents knew, there had been no similar defect in the three preceding generations.

GROUP II.

Case 1.—John N. was one of three cases of congenital deformity of the auricle and external auditory meatus found in this study of nearly 300 cases. The three cases happened to come from the same school. The child had fairly good hearing, so good that the question at once arose as to why he was in a school for the deaf. The answer was that he had been tried twice in regular schools, but did not get along, probably due to the lack of personal supervision which he gets in his class of eight which was impossible in a class of forty.

The vestibular responses were normal.

He had total absence of external auditory meati and a very marked deformity of the auricles.

The Acquired Type of Deafness.

DR. E. W. HAGENS presented four children illustrating the acquired type of deafness. There was no history of middle ear infection or dicharging ears.

The first patient was a girl, aged 18, who had scarlet fever at the age of three. The mother stated that there was no discharge from the ears, but the patient became totally deaf at that time. Her tonsils had been removed in the hope of remedying her hearing. At the present time examination showed total deafness, with no response to the vestibular tests.

The second patient was 13 years of age. At about six years she developed a severe bilateral eye condition and became practically blind. For a time the condition improved, so that at this time she had only a mild opacity in the left eye. The mother stated that within a year after this eye trouble she noticed that the child did not hear well and gradually both ears became deafened. The Wassermann test was 4 plus, and the girl was under antisyphilitic treatment. There was no vestibular response.

Dr. Hagens said he had had five other cases of this type, proved by Wassermann test, with a total loss of vestibular response in all cases.

The third child was 9 years of age. At the age of 5 he was struck by a street car, sustaining a severe injury, which was probably a basal skull fracture. He was unconscious for 48 hours, following which he was unable to hear. There was no history of any discharge from the ear previous to that time, and the parents stated that his hearing was perfect. He now had a total loss of hearing and a total loss of vestibular response.

The fourth patient, a girl, aged 13, gave a history of having had spinal meningitis at the age of 2½ years. The mother stated that the child talked before the onset of the meningitis, but that since then she had been totally deaf. There was no history of any discharge from the ear at any time. The cochlear responses showed total deafness on both sides, and there was a total loss of vestibular response.

DISCUSSION.

Dr. Charles W. Richardson, Washington, D. C., said the survey of the deaf institutions of the country was quite an important piece of work. Efforts have been made for the past five years to bring about this survey. The attempts to engage the aid of any of the national agencies who could furnish financial aid had failed, and those who were interested in the work were quite in despair. About this time two interested people, unknown to each other, consulted the National Research Council. One of these was Dr. Hall of Gallaudet College in Washington and the other was Dr. Richardson, and they were able to interest the Council to such an extent that they were invited to a meeting of the Educational Division. The Council then appointed a committee of five members representing this particular field of investigation to take up the survey. It was found at this meeting that the medical side had not been taken seriously into consideration, and that other surveys had absorbed practically all the funds. The work then was taken up by Dr. Richardson as a voluntary service, and he enlisted the aid of other otologists in the country.

A good deal of admirable work had been done. He paid all honor and respect to Dr. Shambaugh in telling him that his work was a success in every respect. The work of Dr. Rinder of Buffalo had likewise been perfect.

A more intensive study is to be made of these cases. With the plan Dr. Richardson had in mind, he expected to study from 4,000 to 6,000 cases, which will develop a great deal of material in connection with the physical survey of the deaf. Many questions will be solved—the amount of residual hearing, the relation of acquired and congenital deafness as to their reactions to vestibular and cochlear function. The amount of residual hearing is a very important question, as is also the audiometer reactions to both the acquired and congenitally deafened. It will be possible to make Wassermann tests on all patients and to solve many problems in regard to specific infection in these patients. Consanguinity also can be studied.

He agreed with Dr. Shambaugh in the statement that removal of the tonsils is not productive of any improvement in hearing, and believed that in many cases of toxic origin from the tonsil the harm is done before the tonsils are taken out.

Dr. Wendell C. Phillips, New York, complimented Dr. Shambaugh for the very efficient way in which he and his assistants accomplished this difficult piece of work. He also complimented the Society for having men and women who were able to do work of this kind.

This work in New York City has not progressed so far, but will probably be accomplished soon.

He endorsed everything, even the conclusions, that Dr. Shambaugh gave. Regarding the comments on the tonsil operations as a cure for deafness, he was not sure, but perhaps Dr. Richardson was right.

He regretted that Dr. Shambaugh found it necessary to use the word deafness in this connection. A working classification so far as education is concerned, might be deafness without normal speech and deafness with normal speech. It is true that many of the cases of profound deafness in children sufficient to require education in institutions of special schools for the deaf, may be acquired, but this class is extremely limited so far as percentage is concerned. The far larger class mentioned under the heading of acquired deafness with speech was not

included in Dr. Shambaugh's report, and this type of children should always be educated in their regular schools, plus proper preventive treatment and lip reading instruction.

Dr. Robert Sonnenschein called attention to the phase of heredity and cited the work of Edward A. Fay, in which he reported 4,400 families in which one or both parents were deaf. There were 6,700 children, of whom 8.6 per cent were deaf. Where both parents were deaf, 24 per cent of the marriages resulted in deaf children, and over 25 per cent of the children were deaf. Where the parents were related, 45 per cent of the marriages resulted in deaf children, and 30 per cent of the children were deaf. Dr. A. G. Bell, in a survey of 300 marriages of the deaf, found 56 per cent of the children deaf.

Dr. John J. Theobald said he supervised the examination of the ears of 10,000 children in the public schools for the Chicago League for the Hard of Hearing. Three years ago a survey was made by him, under the direction of Dr. Norval H. Pierce. A whisper test was made on every child, and those not hearing in both ears at 20 feet underwent a more detailed functional examination. Usually, inflation sufficed to restore the hearing to normal. Where this failed, careful inspection, by otoscopy and rhinopharyngoscopy was carried out, and the exact nature of any defect recorded. Some of the cases were sent to their physician, others were taken to the free dispensaries by follow-up workers connected with the League. A few were advised to take up lip reading. The large number found to have defective hearing so impressed the League, the Department of Education and the doctors that it was unanimously decided this work must go on.

In seven schools in which they had completed their records 7,538 children had been examined by the teachers. Further detailed examinations revealed the following:

Tubotympanal catarrh (mild)	93
Tubotympanal catarrh (advanced)	66
Chronic suppurative otitis media	42
Nerve degeneration	7
Cerumen (impacted)	63
	271

Thirteen had stapes fixation and 12 were advised to take up lip reading.

The investigation had proved that the examination of all school children's ears is necessary, and this is obviously the duty of the Board of Health and the Board of Education. There are over 300 schools, and it would require too long a time for the League, with its limited resources, to go through them all.

The first duty is to detect the hard of hearing child, and the next, equally important, is to see that he gets the proper care. More time is required to carry out the latter phase of this work than the former, and it is here that the authority of the Department of Education is needed most to overcome the inertia shown by parents of the afflicted child.

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